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EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. XII.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

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PROF. G. HUGUENIN, of Zürich.

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BIOGRAPHICAL SKETCHES OF THE AUTHORS.

Hermann Nothnagel was born on the 28th of September, 1841, in Alt-Lietzegoericke, Brandenburg. From 1859 to 1863 he studied in Berlin. From 1863 to 1864 he was an assistant of Leyden in Koenigsberg and an instructor at the university. From 1868 to 1870 he was an instructor in the University of Berlin. From 1870 to 1872 he was an instructor in the University of Breslau. In 1872 he was called to Freiburg in Baden as professor at the Polyclinic and of materia medica. In 1874 he was called to Jena as professor of clinical medicine and of pathology.

His scientific contributions have been as follows: "Fatty Degeneration of the Organs in Poisoning by Ether and Chloroform." Berliner klin. Woch., 1866, No. 4. "On the Theory of Coughing." Virchow's Archiv, Band 44. "The Vaso-motor Nerves of the Cerebral Vessels." *Ibid.*, Band 40. "The Production of General Convulsions having thei source in the Pons and Medulla Oblongata." *Ibid.*, Band 44. "On the Theory of Clonic Spasm." *Ibid.*, Band 49. "Experimental Researches on the Functions of the Brain." *Ibid.*, 57, 58, 60, 62, 68 Band. "Communications on the Subject of Vascular Neuroses." Berliner klin. Woch., 1867; No. 51. "On the Theory of the Vaso-motor Neuroses." Deutsches Archiv fuer klin. Med., Band 2. "Angina Pectoris Vaso-motoria." *Ibid.*, Band 3. "On the Physiology and Pathology of the Perceptions of Temperature." *Ibid.*, Band 2. "The Nervous Sequelæ of Typhoid Fever." *Ibid.*, Band 9. "Renal Casts in Icterus." *Ibid.*, Band 12. "On Arythmic Motions of the Heart." *Ibid.*, Band 16. "Clinical Communications and Observations on Diseases of the Brain." I. Abth., *Ibid.*, Band 19. "Pain and Disturbances of the Cutaneous Sensibility." Virchow's Archiv, Band 54. "Participation of the Sympathetic in Cerebral Hemiplegia." *Ibid.*, Band 68. "Trophic Disturbances in Neuralgias." Archiv fuer Psychiatrie und Nervenkrankheiten, Band 2. "Central Irradiation of Voluntary Impulse." *Ibid.*, Band 3. "Observations on the Arrest of Reflex Action." *Ibid.*, Band 6. "Arrest of Epileptic Attacks." Berl. klin. Woch., 1876. No. 41. "On the Epileptic Attack." Volkmann's Sammlung klin. Vorträge, No. 39. "On the Diagnosis and Etiology of unilateral Contractions of the Lung." *Ibid.*, No. 66. "On Neuritis in its Diagnostic and Pathological Relation." *Ibid.*, No. 103. "Handbook of Materia Medica." 2 Auflage, 1874, Berlin.

Professor **Obernier** was born December 16, 1839, at Bonn, on the Rhine, where he attended the gymnasium and the university. In 1860 he worked under the supervision of Pflüger; in 1862 he obtained his degree, and in the winter of 1862–3 he passed the state examination. He then took the position of Assistant Physician at the Provincial Lunatic Asylum of Siegburg, and in 1865, after completing his term of military service, he was appointed assistant at the Medical Clinic of Bonn, in which position he remained seven years. Meanwhile, in 1866, he was installed as private instructor of internal medicine, and in the early part of 1870 he was made extraordinary professor. In the early part of 1871 the medical department of St. John's Hospital was placed under his professional charge.

His literary productions have been: "On the Absence of the Initiatory Contraction in the case of the rapidly increasing current," Müller's Archiv, 1861. "De Nervis Uteri," Dissertatio Inauguralis, 1862; an essay that secured a prize from the medical faculty. "Experimental Researches on the Nerves of the Uterus," Bonn, 1865. "The Sputa in Insane Persons," Allgem. Zeitchr. f. Psychiatrie, 1864. "On a Simple Instrument for Determining the Difference in Size of Pupils," Allgem. Zeitchr. f. Psychiatrie, 1864. "Relations between the Size of the Head and Brain Disease," *Ibid.* "A New Method of Measuring the Head," *Ibid.*, 1865. "Communications on Cases," in the Berliner klin. Wochenschrift. "On Derivations of Heat in Febrile Affections," Berliner klin. Wochenschrift, 1867, Nos. 8 and 9. "Sunstroke," with new observations and extensive experiments, and contributions to the normal and morbid development of heat; Bonn, 1867. "Aneurysma Ascendens Aortæ and Insufficiency of the Tricuspid," Deutsches Archiv f. klin. Med., 1869. "Contribution to the Action of Alcohol," Pflüger's Archiv, 1869. Communications (mostly cases) in the Verhandlungen der Niederrheinischer Gesellschaft für Natur und Heilkunde.

Professor **Heubner** was born in 1843 in Mühltroff, in Saxon Voigtland. From his sixth to his tenth year he attended the public school, and afterwards other schools, in Reichenbach in Voigtland, and Grimma in Saxony. From Michaelmas of 1861 to Easter of 1866 he studied medicine in Leipsic. In the summer of 1866 he completed his studies in Vienna. From Michaelmas of 1866 to Michaelmas of 1871 he was clinical assistant at the Leipsic Hospital at the clinic of Wunderlich, and from 1868 he was private instructor at the university of the city. In the year 1873 he was nominated to the position of extraordinary professor, and is now actively engaged in that capacity at the University of Leipsic. He lectures on special pathology and therapy, and holds a propædeutic clinic.

After the cholera epidemic of 1866 he wrote his dissertation "On the Incomplete Reaction after Attacks of Cholera," and he obtained his degree in 1867. He also, among other productions, wrote the following articles: "On Thrombosis of Sinuses;" "On the Action of Camphor;" "On Cerebral Syphilis." In 1871 he wrote on typhoid, dysentery, and some other internal diseases which were observed during the war. All these articles appeared in Wagner's Archiv der Heilkunde.

Quite recently he has published a monograph "On the Syphilitic Diseases of Cerebral Vessels." Leipsic.

Professor **HUGUENIN** was born in the year 1841, in the village of Krauchthal, in the Canton of Berne, Switzerland. He attended the schools of Winterthur and Zürich, then the gymnasium of Zürich, and studied medicine in the latter city, Prague, Berlin, and Vienna from 1861 to 1867. For three years he was assistant in Zürich, at Biermer's clinic (formerly Griesinger's). During 1867 and 1868 he was engaged in the practice of medicine, but in 1869 took up his residence in Vienna, where he worked a long time under Meynert. In 1870 he obtained the position of attending physician to the new Lunatic Asylum in Zürich. From 1872 to 1874 he held the position of director. In 1874 he undertook the charge of the medical clinic of the city.

His literary productions have been: "On the Meibomian Glands of the Conjunctiva." "Marantic Thrombosis of Sinuses: a Contribution to the Pathology of the Acute Exanthemata." "On Facial Paralysis." "On Brain Syphilis." "General Pathology of the Nervous System, Vol. I., containing a Review of the Anatomy of the Brain and Spinal Cord."

Professor E. **HITZIG** was born in Berlin, February 4, 1838. He is the son of the former president of the Royal Prussian Academy of Arts and the Privy Counsellor, F. Hitzig. He attended the French gymnasium of Berlin, then the convent school of Rossleben and the gymnasium of Zuckau, where he graduated in the autumn of 1858. He then studied at the universities of Berlin and Wurtzburg, obtaining his degree in the former city in the autumn of 1862. The following winter he passed the state examination. He then turned his attention chiefly to the study of nervous diseases and allied affections. He took part in the war against France, being absent for eleven months. In the commencement of 1872 he was installed as Private Instructor in Internal Medicine at the University of Berlin, and in March of 1875 he was called to Zürich in Switzerland, as Professor of Mental Diseases, and Director of the Lunatic Asylum of the Canton.

His literary productions include: 1. Researches on the Brain, first published partly in DuBois-Reymond's Archives and partly in the Archives of Psychiatry, and then reproduced in a single volume as an independent work, with new essays. 1874. Hirschwald. 2. Researches on the Brain. New issue. Reichert and DuBois-Reymond's Archives, 1874, 1875, 1876. 3. Studies on Lead Poisoning, 1. Berlin, 1868. 4. Aims and Objects of Psychiatry, Zürich, 1876. 5. On Luxations in the Tarso-metatarsal Articulation, Berl. klin. Woch. On Currents passed transversely through Frogs' Nerves, Pflüger's Archiv, vii. Bd. On a Joint Affection which occurs in severe Hemiplegias; Virchow's Archiv, 48 Band. On the Relative Worth of some Methods of Electrization, Archiv f. Psychiatry, 1872. A number of his minor articles have also appeared in Virchow's Archives, the Berlin klin. Wochenschrift, and the Archiv f. Ohrenheilkunde.

ADDENDUM TO NOTE ON PAGE 474, VOL. VII.

THE formula for the meat solution is contained in an article by Leube, in the Berliner klin. Wochenschrift, and is as follows:

"Take 1,000 grammes of beef, free from fat and bone, put into an earthen or porcelain jar, and add 1,000 c.c. of water, and 20 c.c. of pure hydrochloric acid. Place the jar in a Papin's digester, screw the cover tight, and boil from ten to fifteen hours, stirring occasionally during the first few hours. Then remove the contents of the jar to a mortar, and rub the mass until it has the appearance of an emulsion. Boil again for fifteen or twenty hours without raising the cover of the digester. Add pure potassium carbonate until the mass is nearly neutralized, then evaporate to a pulpy consistence."

The meat solution can be obtained in New York from Mr. F. Hoffmann, apothecary, 797 Sixth Avenue.

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NOTHNAGEL.

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ANÆMIA, HYPERÆMIA,

HEMORRHAGE, THROMBOSIS, AND EMBOLISM

OF

THE BRAIN.

NOTHNAGEL.

INTRODUCTION.

H. Boerhaave, Praelectiones academicae de morbis nervorum. Frankofurti et Lipsiae, 1762.—*S. A. Tissot*, Abhandlung von den Nerven und ihren Krankheiten. Uebers. von Weber. Winterthur und Leipzig, 1781.—*J. B. Morgagni*, De sedibus et causis morborum.— *J. P. Frank*, De Curandis hominum morbis epitome. (Tom. VII.) Viennae, 1821.—*J. Abercrombie*, Pathological and practical researches on diseases of the brain and spinal cord. Edinburgh, 1829.—*Magendie*, Vorlesungen über das Nervensystem und seine Krankheiten. Leipzig, 1841.—*Andral*, Clinique médicale. Paris, 1848. (Vol. 5).—*Andral*, Cours de pathologie interne. Paris, 1848. (Vol. 2.).—*Marshall Hall*, Lectures on the nervous system and its diseases. London, 1836.—*Joseph Frank*, Die Nervenkrankheiten. Leipzig, 1843.—*J. Dietl*, Anatomische Klinik der Gehirnkrankheiten. Wien, 1843. —*Todd*, Clinical lectures on paralysis. London, 1854.—*M. H. Romberg*, Lehrbuch der Nervenkrankheiten. Berlin, 1853.—*Leubuscher*, Pathologie und Therapie der Gehirnkrankheiten. Berlin, 1854.—*Hasse*, Krankheiten des Nervenapparats. In Virchow's Handbuch der spec. Path. u. Ther. Erlangen, 1855. *Durand-Fardel*, Traité clinique et pratique des maladies des vieillards. Paris, 1854.—*Calmeil*, Traité des maladies inflammatoires du cerveau. Paris, 1859.—*Trousseau*, Clinique Médicale de l'Hôtel-Dieu de Paris. Paris, 1865.—*Graves*, Clinical lectures on the practice of medicine. Dublin, 1848.—*Valentin*, Versuch einer physiologischen Pathologie der Nerven. Leipzig und Heidelberg, 1864.—*M. Rosenthal*, Lehrbuch der Nervenkrankheiten. Erlangen, 1870.—*A. Eulenburg*, Lehrbuch der functionellen Nervenkrankheiten. Berlin, 1871.—*Russell Reynolds*, Diseases of the nervous system; by different authors in: A system of medicine. Second edit. London, 1872.— *W. Hammond*, A treatise on the diseases of the nervous system, III. edit. New York, 1873.—*J. M. Charcot*, Leçons sur les maladies du système nerveux. Paris, 1872–73.—Consult also the different text-books on special pathology and therapeutics.

General Considerations on the Circulation of Blood within the Cranial Cavity.

G. Burrows, On disorders of the cerebral circulation; and on the connection between affections of the brain, and diseases of the heart. London, 1846.—*A. Kussmaul* und *A. Tenner*, Untersuchungen über Ursprung und Wesen der fallsuchtartigen Zuckungen bei der Verblutung sowie der Fallsucht überhaupt.

Abdruck aus Moleschott's Untersuchungen. Frankfurt a. M. 1857. 124 S.—*J. A. Ehrmann*, Recherches sur l'anémie cérébrale. Strasbourg, 1858.—*E. Leyden*, Ueber Hirndruck und Hirnbewegungen. Virchow's Archiv. 37. Bd.—*F. Jolly*, Untersuchungen über den Gehirndruck und über die Blutbewegungen im Schädel. Würzburg, 1871.—*F. Pagenstecher*, Experimente und Studien über Gehirndruck. Heidelberg, 1871.—*G. Althann*, Beiträge zur Physiologie und Pathologie der Circulation. Dorpat, 1871.—*Ackermann*, Untersuchungen über den Einfluss der Erstickung auf die Menge des Blutes im Gehirn und in den Lungen. Virchow's Archiv. 15. Bd.—*O. Heubner*, Ernährungsgebiet der Hirnarterien. Ctrlblt. f. d. med. Wiss. 1872. No. 52.—*Duret*, Rech. anat. sur la circulation de l'encéphale. Arch. de Phys. Normale et Pathol. 1874.

It must to-day be regarded as an established fact that the aggregate amount of blood present within the skull is subject to variation, becoming now greater, now less, as the case may be. Monroe (in 1783) maintained the opposite doctrine, namely, that this quantity was fixed, and could not vary so long as the unyielding walls of the skull remained uninjured—unless, indeed, a certain amount of blood, or serum, should escape from its vessels, when a corresponding quantity of that which remained in circulation would be of necessity expelled. This theory of Monroe's had a marked influence upon the clinical views of the day, and this influence was strengthened when the experiments of Kellie[1] were made known, which seemed to show that so long as the skull was left intact, the quantity of blood in the vessels could neither be diminished by bleeding, nor increased by ligature of the cervical veins, or the like. English pathologists in particular, for example, Abercrombie (l. c.), Watson,[2] and others, adopted the supposed results of Kellie's experiments (as a matter of fact the conclusions drawn were not justifiable) as a basis for certain clinical theories; whereas physicians of other countries accepted them only in individual cases (for example, Rochoux and Hammernyk.[3]) The untenability of this view was, however, soon demonstrated in England itself, by Burrows, who showed, by careful experiments, that in the cavity of the skull, just as in other organs, changes, not only in the relative amounts of the arterial and the venous blood, but also in the aggregate quantity, may occur. Donders,[1] and afterwards Kussmaul and Tenner, proved this directly by trepanning the skulls of animals, and then fitting tightly into the opening thus made a piece of glass, through which they were able to observe the changes in fullness of the intracranial vessels. Later investigations, even those of most recent date (Ackermann, Ehrmann, Leyden, Jolly), have lent confirmation to these earlier statements, so that now the belief in the possibility of the occurrence of anæmia and hyperæmia of the brain, which before the time of Monroe had been held without opposition, may be regarded as substantiated by experimental proof. At the same time the circulation within the cranial cavity is in some respects so peculiar, that it is easy to understand how the views upheld by Monroe and Kellie came to be entertained. These peculiarities are due to the fact that the contents of the skull are practically incompressible by the forces present in our organism, while, at the same time, they lie enclosed air-tight in an inelastic capsule. This being so, it is plain that before any new quantity of matter can find its way into the interior of this capsule, a corresponding quantity of its former contents must be displaced, and, conversely, the withdrawal of a portion of the matter formerly present must be made up for by the introduction of a similar quantity from without.

---

[1] Medico-Chirurgical Transac. of Edinb. Vol. I.

[2] Lectures on Med. Med. Gaz. Vol. 27.

[3] Recherches sur l'apoplexie, etc. Paris, 1833.

Of the means for regulating the circulation, the cerebro-spinal fluid constitutes one of the most important (Magendie,[2] Longet,[3] Ecker[4]). When the cerebral vessels become distended, it recedes from the cavity of the skull, to enter again as the distention becomes less.

Further, the investigations of a number of more recent observers have shown that the contents of the perivascular lymph-spaces act in a similar manner (Robin, His, Schwalbe,[5] Manz,[6]

---

[1] Schmidt's Jahrb. 1851. 69. Bd.

[2] Leçons de physiologie.

[3] Anat. et physiologie du système nerveux.

[4] Physiol. Unters. üb. d. Bewegungen des Gehirns und Rückenmarks, etc. Stuttgart, 1843.

[5] Centralbl. f. d. Wissensch. Jahrg. 1868 u. 1869.

[6] Ibid. Jahrg. 1870.

Golgi,[1] Axel Key and Retzius,[2] Mierzejewski,[3] Boll[4]). Although it is true that it is not yet absolutely known in exactly what manner the ventricles, the subdural and subarachnoideal cavities, the perivascular spaces, the vascular plexuses within the ventricles, the sinuses, and the external blood- and lymph-vessels, and lymph-glands of the head and neck are connected together, still there is abundant experimental evidence that connections between them of some sort or other do exist. Thus, according to Schwalbe, injections into the subdural cavity penetrate also into the lymph-glands and vessels of the neck; it is certain that the perivascular spaces and the subarachnoid cavity are united together; and it has been shown that from the latter the injected fluid finds a passage, by way of small open spaces occupied by Pacchionian granulations (Trolard[5]), into the sinuses of the dura mater, indeed, even through the veins of the skull into the vessels of the scalp (Key and Retzius). We can then assume, with Golgi, that, when more blood than usual enters the brain, the perivascular spaces become obliterated, filling up again as the blood recedes. Circumstantial proof of the importance of these spaces for the regulation of the circulation has been given us, as it would seem, by Gaethgens. He found that, through the injection of fresh, defibrinated blood, under high pressure, into the carotid of the horse, the lymph could be made to flow rapidly out of the lymph-vessels of the neck.

The thyroid gland is to be regarded as furnishing a further means for the regulation of the intracranial circulation. Maingien[6] was led, by a series of investigations (partly from the point of view of comparative anatomy), to the conclusion that this action of the thyroid gland lies in the fact that, when under excessive muscular exertion the veins of the neck are compressed, and there is danger of venous congestion in the brain, this gland also becomes swollen, and in its turn compresses the carotids, being itself held firmly against the vertebral column by the contraction of the muscles of the neck. In this manner the danger of further distention of the intracranial vessels, by way of the carotids, is diminished. Guyon[1] confirmed and extended this conclusion by the observation that, during strong muscular exertion, pulsation can no longer be detected in the branches of the external carotid, while that of the radial artery is felt as before.

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[1] Ibid. Jahrg. 1870 u. 1871.

[2] Ibid. Jahrg. 1871.

[3] Ibid. Jahrg. 1872.

[4] Archiv f. Physiatrie u. Nervenkrankh. 4. Bd.

[5] Arch. Génér. de Méd. 1870.

[6] Vide *Longet*, l. c, T. I.

Finally, the importance of the peculiar arrangement of the cerebral sinuses, and the mechanism of the circle of Willis, for the circulation and distribution of the blood, need not be dwelt upon here.

Whether the above-mentioned means for the regulation of the circulation are sufficient, either alone, or together with others as yet undiscovered, to keep the contents of the cranium always the same in aggregate amount, whatever the changes in the fullness of the blood-vessels, is a question to which the clinical study of cerebral anæmia and hyperæmia does not furnish us with an answer ; for, even supposing the contents of the skull, *i. e.*, *the intracranial pressure*, did remain always the same, it is nevertheless evident—as all will admit—that a diminution in quantity of the arterial, or a stasis of the venous blood—in other words, *a reduction in the amount of normally constituted blood*—could not but disturb the functional activity of the brain, in its various departments.

How, then, shall we decide as to whether or not the intracranial pressure (which is, of course, equal to the pressure in the intracranial arteries, minus the resistance afforded by the walls of the vessels, so that in fact only a slight positive pressure is borne by the brain substance itself) can be influenced by changes in the circulation of the blood? The experiments of Leyden and Jolly appear to have answered this question definitely in the affirmative. Venous stasis causes the intracranial pressure to rise ; arterial anæmia causes it to sink. In all cases of cerebral hyperæmia and anæmia, then, two influences—a deficiency of normal blood, and a change in the intracranial pressure—combine to impair the functional activity of the brain. Later in the work we shall discuss them more in detail.

If the above propositions are correct, the different variations in the cerebral circulation must be attended respectively by the following results: 1. Increase in the quantity of arterial blood, by: displacement of the cerebro-spinal fluid and the contents of the perivascular lymph-spaces; or, when the compensation thus made is insufficient, by elevation of the intracranial pressure. 2. Venous stasis by a retardation of the arterial afflux. 3. Diminution in the aggregate quantity of blood, by: an afflux of cerebro-spinal fluid; dilatation and flooding of the lymph-spaces; or, where this is insufficient, by diminution of the intracranial pressure.

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[1] Arch. de Physiol. Normale et Pathol. T. I.

No further argument than a reference to certain well-known anatomical facts is needed to show that the above reasoning applies equally well to the membranes of the brain as to the brain itself.

ANÆMIA OF THE BRAIN.

Besides the two bibliographical lists already given, the reader may consult the following · *Gooch*, Account of some diseases peculiar to women. 1829.—*Ch. Chossat*, Recherches expériment. sur l'inanition. Paris, 1843.—*M. Hall*, Researches principally relative to the morbid effects of the loss of blood. London, 1830. —*Ehrmann*, Des effets produits sur l'encéphale par l'oblitération des vaisseaux artériels qui s'y distribuent. Paris, 1860.—*Lauder Brunton*, On the pathology and treatment of shock and syncope. Read before the Abernethian Society, St. Bartholomew's Hospital, 1874.

Historical Summary.

Anything like exact knowledge of cerebral anæmia and its symptoms belongs to our century alone. There is evidence, it is true, that the affection was not unknown to all the early writers even at the time of Boerhaave; for the express statement frequently occurs, in the latter's excellent academic lectures upon diseases of the nervous system, that a series of cerebral symptoms, such as vertigo, occurring after losses of blood, are due to the "collapsus vasorum in capite." In another place occurs this still more explicit sentence: "Sed apoplexia etiam oritur ab inopia sanguinis boni, qui requiritur, ut corticis [sc., cerebri] vasa impleat." Indeed, one whole chapter treats "de defectu sanguinis rubri in vasis piae matris." But the knowledge of these facts was soon forgotten. Even Peter Frank, who has repeatedly described the symptoms and causes (loss of blood, etc.) of lipothymia, nowhere gives reason to think that he had any exact information with regard to the connection between them. The credit belongs principally to Marshall Hall of having been the first to formulate anew more scientific notions about cerebral anæmia, through his investigations upon the effects of bleeding, and upon the disease which he called hydrencephaloid, and thereby to give an explanation of many cerebral symptoms which had previously been laid to the score of cerebral hyperæmia. Independently of him, but almost at the same time, Abercrombie also was led to adopt the correct view of syncope and apoplexia ex inanitione.

The possession of this knowledge, together with a series of clinical observations, then enabled Andral to write in his work a special chapter upon Cerebral Anæmia. The study of the effects of ligature of the carotids (A. Cooper[1]) was at this time of material assistance in this connection. The many experimental and clinical investigations of later date cannot be singly referred to here, but will be discussed at sufficient length further on. We would mention only the observations of Kussmaul and Tenner as being of special importance.

Etiology.

The differences in the manner of their origin permit the division of cases of anæmia of the brain into a certain number of well-marked groups.

a. This condition may be brought about by influences which act upon the vascular system of the brain alone, or the cerebral anæmia may be combined with a general anæmia, by which the entire organism is affected. Although it is only under the former circumstances that we find the typical form of cerebral anæmia, still we cannot avoid the discussion of its connection with the general condition, because it often happens that, in either case, it is the cerebral symptoms which hold the most prominent place.

b. The cerebral anæmia may affect the entire contents of the cranium, or, more precisely, the entire brain (universal anæmia), or may be limited to certain parts (partial anæmia). The former condition is by far the more common—indeed, the partial anæmia is not only of rare occurrence, but is not always clinically recognizable.

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[1] Guy's Hosp. Reports. Vol. I. London, 1836.

c. Further, the clinical symptoms may differ widely according to whether the anæmia is induced suddenly or gradually, irrespective of its final intensity. Let us now consider the subject more in detail.

Etiology of acute universal anæmia.—There is no clinical condition in which we find acute universal anæmia in so typical a form as is seen when it is experimentally produced by ligature of all the cerebral arteries. This prototype is copied most exactly in cases of great and sudden loss of blood, whether occurring from the veins or from the arteries; arterial losses of blood, however, are more likely to produce the result in a pronounced manner. It also makes but little difference from what part of the body the hemorrhage comes. Thus, for example, cerebral symptoms sometimes follow external injuries, which are accompanied by great losses of blood; they are also observed after profuse hemorrhages from the nose, the stomach, the intestine, and from hemorrhoids; after pneumorrhagia and metrorrhagia, especially post-partum; and after rupture of an internal organ. In former times, though now but rarely, it was often provoked by venesection, of which abundant examples are recorded. In another group of cases *a sudden change in the distribution of the blood in the body at large*, by which a state of hyperæmia was created in some other part, has been observed to cause anæmia of the brain. In this way, as F. Niemeyer points out, the action of Junod's boot may be explained. Clinically, perhaps, this influence is active in cases of "shock," as H. Fischer[1] has endeavored, as it would seem successfully, to prove—reasoning by analogy from the results of well-known experiments of Goltz upon the frog (Klopfversuch). According to this theory there occurs, in cases of shock, a reflex paralysis of the vasomotor nerves, especially the splanchnic, induced at the moment of injury, in consequence of which the blood collects in large quantity in the distended vessels of the abdominal cavity. Obstetricians have long been familiar with the fact that syncope sometimes follows violent labor, even when the loss of blood has been inconsiderable, an occurrence which undoubtedly is due to the rush of an abnormal quantity of blood into the abdominal vessels, which have suddenly been relieved of the great pressure exerted upon them, and to the corresponding withdrawal of blood from the brain. The faintness which sometimes accompanies the too rapid withdrawal of ascitic fluid in paracentesis abdominis is also to be explained in this way.

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[1] Ueber den Shock—Sammlung klinischer Vorträge, herausgegeben von R. Volkmann.

In other cases acute universal cerebral anæmia arises from *insufficient energy of the heart's action.* This often happens in the case of persons convalescent from severe febrile diseases, when they rise suddenly from a lying to a sitting position. The explanation of the occurrence seems apparent, viz., that the weakened muscles of the heart are incapable of driving the necessary quantity of blood to the brain when the body is in an upright position. More difficult to answer was the question whether irritation of the vagus nerve, acting upon the heart, could lead to anæmia of the brain. That this may occur, has, however, been shown by Jolly, who observed that this procedure was followed by a diminution of the cerebral pressure, manifestly due to a diminution of the intracranial blood-pressure, or, in other words, to a diminution in the supply of blood to the brain.

Many forms of fainting occur perhaps in a similar manner, especially those excited by mental impressions. The character of the pulse, which is small, slow, and irregular (inordinatus, P. Frank), in many of these cases of fainting may be adduced in proof of this. We shall speak again later of the anæmia caused by anatomical diseases of the heart.

Again, sudden anæmia of the brain may be brought about by *spasmodic contraction of the cerebral arteries.* The discussion of the epileptic seizure, which would naturally occur in this connection, I will leave to be taken up later; but it is probable that a spasm of the arteries of the brain also underlies many forms of syncope (fainting), as, for example, those excited by emotional causes, by the sight of surgical operations, etc. At the same time, as has just been remarked, it is not impossible that an irritation of the vagus comes into play here.

Perhaps both these causes act sometimes in conjunction, and perhaps—and this is specially worthy of attention—the influence of the depressor nerve is also to be taken into account. Whether cases like that, for example, reported by Hovell, where a healthy man suddenly died from the emotion excited by the news that a bill, in which he was strongly interested, had failed to pass, are to be explained by the assumption of arterial spasm, or of a kind of shock, or of a powerful irritation of the vagus, must be left undecided. Further, cerebral anæmia can be excited reflectively by the irritation of the sensitive nerves (pain), as I have experimentally proved.[1]

The syncope which sometimes accompanies catheterization is manifestly to be referred also to the irritation of the vasomotor centres, with the production of cerebral anæmia as a result.

Finally, acute, universal anæmia may be caused by the *sudden introduction into the cavity of the cranium of a foreign mass capable of exerting compression.* Clinically speaking, only a large effusion of blood could produce this effect; but, since the circumstances under which this occurs are very complex, and the symptoms by no means to be entirely referred to cerebral anæmia, we will leave this point to be considered in connection with cerebral hemorrhage.

We will pass by the cerebral anæmia from *asphyxia*, treated of by Ackermann, since that is not only of secondary interest in this connection, and, moreover, bound up clinically with a number of other conditions, but, indeed, according to the recent investigations of Jolly, makes its appearance only at the moment of death. The cerebral symptoms, also, excited by certain *poisons* and laid to the score of cerebral anæmia, we will omit to speak of, because the conditions of its occurrence are by no means simple and not yet sufficiently understood. The so-called *gastric vertigo*, which is supposed to be sometimes connected with anæmia of the cranial cavity, we shall speak of later, in connection with hyperæmia of the brain.

Etiology of universal cerebral anæmia of gradual develop-

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[1] Virchow's Archiv. 40. Bd. Vide also the Exp. of Jolly and Riegel, and of Krauspe, ibid. Bd. 52 and 59.

ment.—Among the influences concerned here, the *withdrawal of considerable quantities* of the nutrient fluids of the body, to speak in general terms, plays the most important part. Marshall Hall was the first to point out the fact—of immense therapeutical significance—that many cerebral symptoms (coma, convulsions, etc.), especially if occurring in childhood, which up to that time had been referred to inflammation or hyperæmia of the brain, are dependent upon cerebral anæmia alone. To the group of symptoms having this common origin he gave the name of *hydrocephaloid* (also hydrencephaloid), from their resemblance to the symptoms of true hydrocephalus. Hall showed that, with children, not only repeated losses of blood, but also attacks of cholera infantum, may produce this condition; and suppuration also, but more rarely.

Too long continued lactation may act in a similar manner with women. The effect of insufficient nourishment ranks next in importance to that of loss of the nutrient fluids. The greater this insufficiency, the more extreme is the cerebral anæmia with its symptoms. (It is, of course, more than probable that in these cases the conditions are by no means simple, that not cerebral anæmia alone, but also a direct disturbance of the nutrition of the nervous system, is to be taken into account.) To this cause are to be referred the cerebral symptoms observed to attend starvation, as, for example, among shipwrecked sailors. When, to insufficiency of nourishment, even if not absolute, is added a direct increase of tissue-consumption, the result is naturally more marked. The most prominent example of this is seen during recovery from acute febrile diseases. The longer and more violent the fever, and the more extreme the refusal or withdrawal of nutrient matter, the sooner cerebral anæmia becomes developed, such as occurs in typhoid, scarlet fever, measles. I would call special attention, however, to the fact that this happens most frequently with those febrile diseases which terminate with a regular crisis, and are followed by a well-marked epicritical state of collapse. Among these are particularly to be mentioned pneumonia and relapsing fever (Fraentzel).[1] It was, to be sure, recognized by the excellent observer, Graves, that the cerebral symptoms making their appearance towards the end of, and in convalescence from, typhoid fevers generally require a stimulant and tonic treatment; but, nevertheless, even he considered cerebral hyperæmia to be their cause. The condition was more correctly understood by Andral. In phthisis, especially towards the end of life, similar influences are at work, particularly if the process has been of long duration and if there has been much fever and anorexia. Finally, chlorosis, regarded in general terms as a condition of inanition, can be ranked with the diseases mentioned; certain of its symptoms are unquestionably referable to cerebral anæmia.

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[1] Ueber Krisen und Delirien bei Febris recurrens. Virchow's Archiv. Bd. 49.

Heart diseases, also, play a part in the etiology of this disease. It has already been remarked that they may lead to an acute anæmia; but in those cases where there is diminished functional capability of the muscles of the heart, a less degree of cerebral anæmia is always present, and acute symptoms make their appearance only when the affection reaches a certain degree of intensity or becomes suddenly more violent than before. Above all, the fatty heart is to be mentioned in this connection, which has been considered, especially by Stokes, as so common a cause of cerebral anæmia, that, if the symptoms of the latter affection are present, there is reason to suspect the existence of the heart trouble. Many valvular lesions also exert an analogous influence upon the cerebral circulation, especially those of the aorta, when no longer compensated for by the hypertrophy of the left ventricle.

Finally, *the introduction of foreign matter into the cranial cavity* can also give rise to a more or less progressive form of anæmia; such are the inflammatory exudations in meningitis, or the fluid which transudes in œdema of the brain. The conditions which lead to these processes, also, are quite complicated, and the symptoms which attend them are not always to be referred exclusively to the anæmia.

Etiology of partial, circumscribed, cerebral anæmia.—Attention was called, at the outset, to the fact that the peculiar arrangement of the vessels forming the circle of Willis exerts a decided influence in diminishing the tendency to partial anæmia, especially of an entire half of the brain. The recent investigations of Heubner and Duret have moreover shown that in the so-called ganglia (nucleus lenticularis, corpus striatum, thalamus opticus) the distribution of the vessels is such that ischæmia can be produced much more readily there than in the cortex, where the manifold anastomoses place its occurrence almost out of the question. We shall speak again of this point in connection with cerebral hemorrhage.

Unilateral anæmia is seen in its most typical form after ligature of the carotid on one side, especially in cases where failure of development, or an impervious condition of the communicating arteries of the circle of Willis, renders the communication between the two halves of the brain imperfect.

These irregularities were found by Ehrmann in nineteen or twenty per cent. of the cases, in a number of bodies chosen at random; and, at the same time, the records of ligature of the carotid show that well-marked and more or less permanent cerebral symptoms occurred in twenty-one per cent. of all the cases—most certainly a remarkable coincidence. Of course, when "cerebral symptoms" are spoken of in these statistical statements, as, for example, in the valuable records of Pilz,[1] where such symptoms are reported as occurring in only one hundred and sixty-five out of five hundred and twenty cases, it is only the severer symptoms that are meant. Slight, and passing disturbances, due to the temporary anæmia which is always produced, seldom fail to occur—never, in fact, unless the circulation through the carotid is only gradually cut off, as in thrombosis.

Emboli swept into the cerebral arteries, or autochthonous thrombi, set up more circumscribed forms of anæmia; but, as will be shown later, these are not cases of anæmia pure and simple. Finally, *local spasm of the vessels* may be mentioned as a cause of circumscribed anæmia. So far as we know, this may occur under either of two conditions: in certain forms of hemicrania; and in that modification of the epileptic seizure known as petit mal. Both of these affections will be described in another part of this work.

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[1] Langenbeck's Arch. f. klin. Chirurgie. IX. Bd.

In conclusion, certain *general conditions*, which seem to favor the occurrence of the symptoms of cerebral anæmia, deserve a moment's notice.

Thus these symptoms appear more readily, in greater intensity, with children than with adults; and of the latter it is especially the debilitated, pale, and so-called nervous persons, particularly women, that are pre-eminently subject to them.

Aged persons, also, as I have often noticed, are liable to be affected by syncope and similar affections, on comparatively slight provocation, such as a trifling attack of diarrhœa.

Pathological Anatomy.

Anæmia of the brain is characterized anatomically by the pale color of the organ. This is especially evident in the gray substance, which is sometimes absolutely decolorized, and has a dirty grayish appearance.

Even in the white substance the usual blood-points are absent, and its surface looks therefore whiter than is normal. As to the consistency of the brain-substance and its moistness, they differ with the cause of the anæmia. If the latter is due to a transudation of fluid, the cut surface of the brain will be moist and shining, although pale—its consistency less than normal; if to the pressure of a quantity of extravasated blood, the tissue of the organ will be found rather dry and tough.

The ventricles often contain more fluid than usual. The perivascular spaces are, according to Golgi, enlarged, even when there is œdema of the brain-substance.

Also of interest is the fact, ascertained by Chossat in his inanition experiments on animals, that, in the general anæmia due to inanition, the brain loses almost nothing of its weight, while the muscles lose about forty-four per cent.; the liver, spleen, and the blood about sixty per cent.; and the adipose tissue even as much as ninety per cent.

As in the cerebral substance, so also in the membranes, the amount of blood in the vessels is more or less diminished, although there is almost always a certain quantity in the larger veins and in the sinuses. It is a mistake to think that the meningeal vessels become absolutely empty in anæmia, even after death from loss of blood—in fact, the contrast between their appearance and that of the brain itself is sometimes very striking.

Experimental Investigations.

In order to get at a better understanding of the clinical phenomena attending cerebral anæmia, and, in general, to attain an exact acquaintance with its symptomatology, we will look briefly at the recorded testimony of experiments upon men and upon animals.

Among the former the observations of Jacobi,[1] Kussmaul and Tenner, and Schiff,[2] are of especial importance. In consequence of pressure upon one carotid artery, there is found to be, first, a slight indistinctness of vision ; then, after two or three seconds, a prickling sensation is felt, first in one half of the face, then over the whole of one side of the trunk, and even on the opposite side of the body from the artery. Very soon the general sensibility of all the parts mentioned becomes indistinct, the sense of touch impaired, the control of muscular movements less perfect. Trembling and convulsive twitching may also occur ; but after about one minute, in spite of continuance of the compression, all these symptoms cease.

The compression of both carotids is followed by darkening of the field of vision, while the pupils sometimes contract at first, but always become finally dilated.

The respiration becomes slow, deep, and sighing, accompanied by a sense of oppression about the thorax. Then follows dizziness, drowsiness, staggering, and finally loss of consciousness.

If the compression is still persisted in, motions of choking and vomiting take place, with universal muscular twitching, as Kussmaul and Tenner witnessed in the cases of two rather anæmic persons.

As for the exceedingly numerous *experiments upon animals*

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[1] *M. Jacobi*, Die Seelenstörungen in ihren Beziehungen zur Heilkunde.

[2] Lehrbuch der Physiologie.

(A. Cooper, Kussmaul and Tenner, Ehrmann, Jolly, and many others), we must limit ourselves to the bare mention of their more important features.

If the circulation through both carotid and vertebral arteries is suddenly cut off, the nostrils, eyelids, and pupils contract, soon to dilate again, and the jaw also is closed ; the eyeballs roll upwards and outwards ; the respiration becomes at first labored and short, then slow and deep ; the muscles of the neck give way, and the animal collapses, and falls in syncope. Then follow intense, general convulsions, with loss of consciousness. If the blood be then allowed to flow again, the normal condition after a time returns. Analogous symptoms attend bleeding to death.

That these symptoms depend upon the failure of the arterial blood-supply to the brain has been ascertained beyond a doubt. It is an important fact that when, in either of these experiments, the animals are previously in a very feeble state, or when the bleeding takes place only slowly, the epileptiform convulsions fail to occur, and death supervenes upon simple syncope.

Ligature of the carotids alone causes only a slight, temporary quickening of the respiration and the pulse (A. Cooper, Schiff).

The apparent difference between the results of this operation as performed on man and on animals is explicable, in the first place, on the ground that the carotids do not stand in the same important position with relation to the cerebral circulation, as compared with the vertebrals in animals, that they do in man, and, further, from the lack of the power of conscious expression on the part of the former (comp. above).

Ligature of the vertebrals alone (Cooper, Schiff) makes the respiration temporarily quick and labored, and causes a certain stiffness and weakness in the movements of the limbs.

From the experiments of Chossat we learn the interesting fact that animals exposed to starvation become drowsy, close the eyes, and finally die in a state of coma without convulsions. It is only in case death is delayed until the animals have been revived again by warmth, in the last stages of the process, and until pulse and respiration have become stronger, that it is attended with convulsions.

Whether the fact, observed by A. Durham, that cerebral anæmia (paleness of the surface) is present during sleep, can be turned to account in studying the symptomatology of the former condition is very doubtful, since it is not perfectly apparent which state is here the cause and which the effect of the other.

The view that cerebral anæmia may be due to arterial spasm is supported by my experiments, which show that this condition may be brought about through the influence of reflected irritation, as is well known to occur also in various parts of the body besides the brain.

Finally, the experiments of Navalichin,[1] and those of Mosso,[2] are to be mentioned, both of whom found that increased rapidity of the pulse and increase of the general blood-pressure were the direct result of cutting off the arterial blood-supply of the brain. We shall refer again later to the different interpretations which the authors put upon these observations.

Symptomatology.

The symptoms of cerebral anæmia vary very much in their nature, according to the intensity of the disease and the rapidity of its onset.

We will begin by sketching some typical modes of manifestation of the affection.

We will suppose that a person unused to such scenes attends a surgical operation. Not absolutely suddenly, but gradually, after the lapse of a few minutes, he feels somewhat oppressed for breath, is inclined to gape, cannot continue to attend and observe as closely as before, commonly breathes somewhat deeper. Shortly after, his face becomes very pale, the sense of oppression increases and becomes intense, and is accompanied with dizziness and a feeling of general relaxation; he can scarcely keep himself upon his feet. A cold perspiration breaks out on the face, and sometimes on the entire body; the ears begin to ring ; he is nauseated and inclined to vomit. His field of vision becomes progressively more obscure and veiled ; the surrounding voices fall on his ear as a senseless medley of sound. The pulse, though regular, is small and of diminished tension. Can he leave the room at this moment, either led by another or stumbling along alone, and lie or sit down quietly for a time, all these symptoms soon pass away, leaving only a certain paleness of the face ; if not, he soons falls insensible, without a sound, to the floor, the pulse remaining as described, the respiration quiet, the face very pale. After a few moments he begins to recover himself.

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[1] Centralblatt f. d. med. Wiss. Jahrg. 1870.

[2] *A. Mosso*, l'Imparziale. 1872. S. A.

This is the picture of a simple *fainting-fit.* Three varieties of it are described ; varying in the degree of intensity of the symptoms, that range from a feeling of general weakness to absolute coma, under the names of *eclysis*, *lipothymia*, *syncope.*

In a woman who has just given birth to a child—the labor up to that point having been a natural one—a profuse metrorrhagia occurs when the uterus fails to contract with sufficient force. If energetic measures are not adopted, all the symptoms just described as characterizing inanition will be developed, and then will follow—either at the moment of fainting or afterwards—convulsions of the most violent character, resembling in every respect epileptic convulsions.

Very different are the symptoms of anæmia which occur in connection with the so-called *hydrencephaloid*, made classic by the writings of Hall, and, afterwards, of Gooch and Abercrombie.

For example, take the case of a child of one year old who has been suffering a number of days from a severe attack of cholera infantum. It lies in a condition of excessive exhaustion, the eyes closed during the greater part of the time, retracted in their sockets, and surrounded by dark rings. It inclines almost constantly to fall into a sleep, which is like that of insomnia, or, to say the least, restless, and preceded each time by a season of fretting and crying. Face, as well as hands and feet, are pale and cold ; the pulse is moderately rapid, regular, compressible ; the respiration is about 20, and regular. The fontanelle is depressed ; the pupils are contracted ; there is often strabismus, sometimes contraction of the muscles at the nape of the neck.

Gradually the somnolence gives place to complete coma, during which the pupils are dilated, and may react as little to light as the cornea to touch; in this stage the respiration is slower than normal. Death may supervene upon the coma, or, under appropriate treatment, recovery may still take place.

Still other features characterize the cerebral anæmia of *chlorosis*, others the post-febrile, and still others the local variety of the disease. We regard it as advisable, however, in order that constant repetitions may be avoided, to give rather an analysis of the different symptoms, taken each by itself, than descriptions of the many and various combinations in which they clinically occur.

Analysis of the different Symptoms.

1. *Sensorial disturbances.*—Disturbances in the higher functions of the brain are never wanting in cerebral anæmia, although they may vary exceedingly in character and intensity, according to the character of the special cases. Sometimes no other symptoms besides them are present, and, when the cerebral anæmia is at all marked, they precede all the rest in the series, unless it may be disturbances of respiration. The cerebrum, then, and especially the cortex cerebri, is evidently, of all organs, the one most easily disturbed in its functions, in consequence of the lack of arterial blood.

This functional disturbance may manifest itself, now through phenomena of irritation, now through those of depression. A certain *mental torpor* is frequently characteristic of anæmic, debilitated individuals. Cases are occasionally reported which give direct evidence of the degree to which the activity of the mind is dependent upon the blood-supply to the brain; the patient, not being capable of intellectual labor when in the upright, but only when in the horizontal position. This same mental dulness is to be seen also after losses of blood of some magnitude; chlorotic young women show it in particular. Hand in hand with this goes an unusual *tendency to drowsiness*, such as is also met with in acute anæmia, but especially in cases of hydrencephaloid. Whether the sense of *dizziness*, which is one of the commonest symptoms of the lighter degrees of cerebral anæmia, is, strictly speaking, a cerebral symptom or not, it is difficult to decide (Comp. the chap. on "Dizziness," in a later volume.) If the supply of blood is still further diminished, the sensitiveness to outward impressions becomes blunted and lost, until the patient falls into a condition of *somnolence*, and finally complete *coma*. Absolute loss of consciousness is found only in connection with the more or most extreme degrees of anæmia, and it seems to me that the anæmia must come on with a certain suddenness, or at least must suddenly increase in intensity, before that condition can occur; when taking place under these latter circumstances, this loss of consciousness was called by the older writers "apoplexia ex inanitione," as, indeed, Abercrombie and Marshall Hall remark.

On the other hand, in many cases, especially those of chronic development (chlorosis, etc.), well-marked *mental irritability* is seen, often associated with fretfulness, restlessness, and unquiet sleep disturbed by dreams, and with abnormal irritability of the organs of special sense, especially the eye and ear. Further, in contrast to the sopor and coma above mentioned, cerebral anæmia may be characterized by *delirium*, and it is as yet impossible to tell why it is that in one case this state, in another its opposite is induced. At the same time, it is an undeniable fact that cerebral anæmia in general much more often gives rise to the so-called symptoms of depression than to the reverse conditions. The delirium, when it occurs, may come on either before or after loss of consciousness, in acute as well as chronic anæmia.

It is most common under the following conditions: In the first place, it comes on as a consequence of hemorrhage; here it seldom occurs as a primary symptom, following on simple restlessness, dizziness, ringing of the ears, but, as a rule, only as a sequence of syncope, and then oftener with robust than with feeble individuals, though it is seen with the latter.

On the approach of a fatal result the delirium gives place to coma. Hall looked upon this delirium as a symptom of excessive reaction, which is of course a term only without meaning. Delirium occurs but rarely, as compared with syncope, immediately after great losses of blood; it is more common as a consequence of starvation, especially during and after the acute febrile diseases, and gets under these circumstances the name of "*delirium of inanition.*"

This expression strikes me as being more appropriate than the name "delirium of collapse," which was chosen by H. Weber. The former is of wider application; and, moreover, no fundamental distinction, either as regards their nature or their outward features, is to be drawn between the delirium of post-febrile collapse and that which is observed in phthisis and similar affections. The conditions under which delirium occurs by preference have already been touched upon; next to them are to be mentioned the cases where mental disturbances appear in persons who have been for a long time suffering from hunger, or whose food has been suddenly reduced in quantity, so as to be relatively insufficient, as was sometimes formerly the case in prisons. The delirium of febrile diseases appears usually after the crisis, in the stadium decrementi, or during convalescence. Whether, when it occurs during the period of marked elevation of temperature, it is to be regarded as of anæmic origin is a difficult question to decide, yet not always impossible, especially when, besides the fever, all the other symptoms (feebleness of the pulse, wasting and paleness, trembling) point to a state of general depression. The rapidity of development of the condition varies; it may increase slowly to its maximum, or may break out suddenly, sanity passing into insanity almost without discoverable gradation. Sometimes the patients wake in the morning with signs of the mental disturbance unexpectedly present.

The delirium of cerebral anæmia has, in almost every case, the same character and similar features. Almost without exception the patients are excited and sometimes maniacal. Hallucinations, especially of sight and hearing, are prominent features of the attack; delusions of persecution may be present in typical form.

In spite of the excitement, however, the strongest of the delusions have almost always an undertone of sadness, and the collective symptoms are therefore commonly those of the so-called melancholia agitans. The duration of the condition is not always the same: it is generally brief, giving place, after one or more days, or even hours, in light cases and under appropriate treatment, to the normal state; but it sometimes lasts for weeks.

Occasionally the mental disturbance continues, and passes over into *permanent insanity.* Upon this point we cannot enter here at greater length. The delirium which has been sometimes observed after ligature of the carotid is generally to be explained on the assumption of deeper anatomical lesions, such as meningitis, cerebral abscess, etc.

2. *Disturbances of the organs of special sense.*—Of these organs only the optic and auditory are ever affected; at least no observations, clinical or experimental, are recorded, showing impairment of the functions of smell and taste.

Ringing or buzzing in the ears is one of the commonest symptoms of even the slighter degrees of anæmia. Whether the partial deafness that attends incomplete syncope (eclysis) is due to a special affection of the auditory nerve, or to the weakened mental power of perception, is still a matter of doubt. That the former supposition is not inadmissible seems to be proved by Abercrombie's oft-quoted case, that of an excessively debilitated and wasted patient, who was deaf so long as he was in the upright position, but could hear perfectly while lying down or whenever he bent himself forward so that his face became reddened.

The ringing in the ears may be paralleled by an analogous affection of the optical apparatus, viz., the appearance of specks before the eyes; this may turn into, or be connected with, a darkening of the field of vision and blurring of the outline of objects within it. Here again it must remain uncertain whether the optic nerve itself or the central power of perception is at fault. I have learned, orally, from my former colleague, Prof. Manz, that in the many ophthalmological examinations which he had made of chlorotic and otherwise anæmic persons, he had found at most a general and slight paleness of the fundus, but no marked changes in the size of the vessels.

Total *amaurosis* is a tolerably rare symptom. Most of the examples of it have been observed after hemorrhage from gastric ulcers, more rarely after severe bleeding from other causes, and more rarely still in connection with inanition. The diminution in the power of vision increased in these cases in the course of several (generally from eight to twelve) days after the hemorrhage to complete and commonly permanent blindness.

Sometimes during this period before the blindness comes on, pain in the head is present, sometimes no disturbance of any sort, the amaurosis becoming developed suddenly in the course of a few hours.

In the cases observed by A. von Graefe there were no noticeable changes immediately after the hemorrhage, but a little later atrophy of the optic papilla was found; and he is inclined to assume that the whole trouble is due to a sudden attack of retro-bulbar neuritis. Samelsohn found in one case at first spots of exudation of gray color in the retina, and afterwards a large mass of extravasated blood, which, he thinks, found its way thither from the sheath of the optic nerve, where it had probably been originally poured out (cited in the Allg. med. Centralzeitung, 1874, No. 40).

3. *Disorders in the sphere of the sensitive nerves are more rare.*—Headache is, to be sure, pretty common; but even this symptom belongs almost exclusively to those cases where the anæmia is of slow development. Thus chlorotic women suffer from it, and those persons, too, who have lost large quantities of blood or are otherwise debilitated. It is seldom present in great severity, but, on the other hand, may persist with obstinacy; it is generally bilateral and felt over the entire head.

Sometimes, to be sure, it is unilateral, but not necessarily to be classified with the true hemicrania vasomotoria on this account. As a rule, it is accompanied with irritability of temper, or, on the other hand, with mental dulness, sometimes with dizziness and nausea.

When the anæmia and its accompanying symptoms are of acute origin, headache is rarely present. In cases of ligature of the carotid alone is it observed with any frequency; and even of these cases only those really ought to be taken in evidence in which the headache made its appearance immediately after the operation on the same side of the head, and soon disappeared again (nineteen times out of the great numbers collected by Pilz).

Other sensory disorders besides those mentioned may be induced by ligature of the carotid, as Schiff's experiments upon unilateral compression of the carotid show. These have been observed, it is true, but seldom ; still one reason for this may lie in the fact that, in the superficial examinations which are usually made, all but the more prominent changes in sensibility escape unnoticed ; among these anæsthesia of the opposite half of the body has been occasionally reported.

4. *Disorders in the sphere of the motor nerves* are very common, and they also can manifest themselves by symptoms of irritation or of paresis.

In the latter class the sense of general weakness is to be reckoned, the inability to maintain the upright position, which is an almost constant attendant of the lesser grades of acute cerebral anæmia, and often the precursor of syncope. It is analogous to the paresis, the feeling of excessive weakness, which is seen in the extremities of the opposite side of the body in case of ligature or compression of one of the carotid arteries.

This weakness may either be temporary merely, or it may give place to the general paralysis of coma. Complete paralysis, *without* coma, does not belong among the symptoms of *universal* cerebral anæmia (although, as is well known, it occurs frequently in spinal anæmia) ; but, on the other hand, it has been observed pretty often—in eight per cent. of Pilz's cases—in consequence of ligature of the carotid. The distribution of the paralysis in these cases varies ; the upper extremity alone, or the lower (of course, on the side of the body opposite to that of the ligature) has been found affected, or both together, sometimes with the facial nerve as well. The paralysis of the latter was in a few cases crossed with that of the limbs—*i. e.*, on the same side with the ligature.

When death is not caused by complications of some sort, and when the circle of Willis is normally developed, so as to permit of the establishment of compensatory circulation, the paralysis may disappear after a few days.

Among the most interesting results of cerebral anæmia belong the *motor symptoms of irritative origin ;* and the credit of having investigated the relationship between them belongs to Kussmaul.

If it must be confessed that all these symptoms are not seen together, with the same completeness and exactness at the bedside as in the laboratory, nevertheless, they may all, under fitting conditions, be observed.

It has practically been found that, leaving epileptic attacks out of consideration, it is almost exclusively one certain form of cerebral anæmia which causes the motor symptoms of irritative origin—and that is the anæmia due to hemorrhage; and there are, furthermore, three additional factors which are essential to its production: the aggregate loss of blood must be considerable; it must occur all at once, or undergo a sudden increase; and the patient must not have been previously in excessively debilitated condition.

The primary consequences of the anæmia occurring under these circumstances, the contraction of the iris, etc., have clinically been hitherto but too little regarded. The same is true of localized muscular twitchings.

The largest share of attention has always been given to the familiar *general convulsions*, whose distinctive features are so well given in the term "epileptiform" that we need not describe them further. These convulsions are always associated with loss of consciousness. According to the degree of the anæmia, the patients may either recover or may die in coma, or during the convulsions themselves.

When the anæmia is induced gradually, it does not lead to convulsions—not even in children with hydrencephaloid, in spite of their natural liability to such outbreaks (at the most, spasmodic contractions of isolated parts occur, such as the muscles of the eyes (strabismus) or of the neck). The same is true in the other forms of chronic anæmia, depending upon reasons which will be discussed later.

In epilepsy the conditions are more complicated, as will be shown elsewhere. In the case of meningitis, of cerebral tumors, and cerebral œdema, the influence of increased intracranial pressure comes into play, in addition to the anæmia, as a factor in the production of convulsions.

On the other hand, convulsions of the opposite side of the body, as well as indeed general convulsions, sometimes follow ligature of one carotid artery, although but seldom—in sixteen out of the five hundred and twenty cases recorded by Pilz. Yet even this proportion must appear strikingly large when we reflect that the convulsions are initiated in the pons Varolii, which is nourished by the basilar artery, and should therefore, strictly speaking, not be affected at all by ligature of the carotid. Probably in such cases anomalous arrangements of the blood-vessels exist.

5. *Disorders of the organs of respiration, circulation, and digestion.*—It has already been noted above that the first symptom of acute anæmia, sometimes even preceding the sensory disturbances, consists in a change in the character of the respiration—an indication that the respiratory centre is more susceptible than even the cortex cerebri to changes in circulation of the blood. The respiration becomes at first deep, sighing, sometimes slow. One striking symptom is a tendency to frequent gaping and a sense of suffocation, of want of air, which is very often to be observed in commencing lipothymia.

During complete loss of consciousness the respiration is generally weak and regular, interrupted now and then by deep sighs. If, as occasionally happens, the coma tends to result fatally, it becomes noisy, deep, and finally intermittent.

The character of the pulse varies according to the (etiological) form of the anæmia. The experimental results obtained by Navalichin and Mosso would lead us to expect to find the arterial tension increased. That this is not, in fact, observed clinically—the artery being found either to offer a normal degree of resistance, or even to be abnormally compressible—is plainly due to the fact that the influences which induce anæmia of the brain generally diminish at the same time the energy of the heart's action, either directly, or indirectly by diminishing the mass of the blood (hemorrhage, inanition). The rapidity of the pulse likewise varies with the etiological form of the anæmia; in simple syncope it is usually normal; if general anæmia coexists with cerebral, it is commonly increased; while it is lessened if the intracranial blood-pressure is above the normal.

In the latter case the pulse may also become irregular, which only exceptionally happens in simple anæmia.

One of the most constant symptoms of cerebral anæmia, and one which in many cases settles the diagnosis, and is very often the first outward sign of the disorder, is *paleness of the face.* Where universal anæmia or inanition exists, they would, of course, of themselves give rise to this condition ; but even in the acute forms of the disease in question it is rarely absent, and indeed is, of all the symptoms, one of the first to appear and last to vanish.

It may often become extreme, and be attended with coldness of the cheeks, ears, and nose, and sometimes—in case the activity of the heart is diminished—with analogous conditions of the entire surface of the body. The forehead also is in common syncope often covered with perspiration. Occasionally a regular chill occurs, and quite frequently horripilation ; this may be due at times to an irritation of the vaso-motor centre attendant on the cerebral anæmia ; but certainly, as a rule, the horripilation and the cerebral anæmia, when they occur together, are coincident effects of one cause (vascular spasm).

Finally, certain symptoms of gastric disturbance, *nausea, movements of vomiting*, and even *vomiting* itself, are to be mentioned. The former are of frequent occurrence ; the latter is more rarely seen.

Setting aside the cases of chronic anæmia where increase of the intracranial pressure is present, such as may of itself give rise to nausea and vomiting, these symptoms are met with exclusively in acute forms of the disease (syncope, hemorrhage, etc.), or, at the least, in acute exacerbations.

When occurring in connection with loss of consciousness, they usually precede it, but may also make their appearance after the patient has again come to himself.

Pathology.

What is the pathological action of this anæmia ? Through what process does it produce the effects that have been described ?

It seems possible that the ill-effects of a diminution of the blood-supply to the brain should be threefold, consisting in: 1. Diminution of the intracranial pressure. 2. Reduction of the gross amount of arterial blood below the point necessary for the maintenance of the cerebral functions in their normal state. 3. Perhaps a qualitative change in the blood.

Burrows believed the syncope to be only indirectly due to the reduction in the total amount of the blood supply, and directly to a falling off of the pressure exerted by the blood-vessels upon the cerebral substance; and although it may well be that this notion is too narrow, yet it is equally unreasonable to set this influence entirely aside, as is almost universally done at the present day. That the intracranial pressure can be diminished is certain; and it seems by no means improbable that, when this diminution occurs suddenly, disturbances of the cerebral functions should follow. Unfortunately it has not yet become possible to test the theory experimentally.

As regards the other two influences set in action by anæmia, we have learned to understand them better through the numerous investigations concerning the physiology of respiration.

It is true that the physiological reaction of the respiratory centre may differ somewhat in character from that of other cerebral centres, but there can, nevertheless, be no doubt that the essential nature of the chemical or physical processes in the ganglion-cells must be in both cases the same. To discuss at greater length the vexing question, as to the mode in which the excitation of the respiratory centre is brought about, would be out of place here.

Clinical observation has shown that the different regions of the brain are not all equally affected when their blood-supply becomes deficient, but some more easily than others.

The respiratory centre is one of the first to respond to this morbid influence; but, on the other hand, it is only in the extreme degrees of anæmia that this centre becomes absolutely paralyzed.

Together with, or even at times before, the respiration, the functions of the cortex cerebri, the seat of the psychical processes, suffer in consequence of deficient blood-supply. Numerous and varied as the sensorial disturbances are, it is, nevertheless, utterly impossible to trace, even indistinctly, the existence of any clinical law governing the order of their occurrence. From comparing a number of clinical observations, I should say, on the whole, that the occurrence of delirium indicates a less degree of anæmia than that of coma and sopor; further, that the rapidity of onset of the anæmia is of importance in determining the character of the mental symptoms; if it comes on slowly, the symptoms of irritation will predominate, if rapidly, those of depression.

The combination in varying degrees of these two factors must of course, though in a manner which is unknown, cause many and varied phenomena of irritation and depression to appear.

We have spoken above of the disturbances of special sense. As to what is the seat of the changes which give rise to pain in the head, it is hardly safe to express a definite opinion; it is, however, not improbably the dura.

Somewhat more advanced—thanks especially to Kussmaul and Tenner—is our knowledge of the motor irritative phenomena, the convulsions. The seat of the changes which underlie them is to be sought in the medulla oblongata, or rather, according to my experiments, in the pons Varolii; the ganglion-cells concerned are acted upon by the influences included under the head of anæmia, in the same manner as are those of the neighboring respiratory centre.

Experiment and clinical experience coincide in teaching that the anæmia must have reached a high pitch before convulsions are excited, for which reason they never occur without coma; further, the onset of the anæmia must be rapid, and the patient not, previously, too much enfeebled, which explains the absence of convulsions in hydrencephaloid. The fact that the convulsions imply an irritation of the pons makes clear why it is that they are almost always wanting in case of anæmia of the brain alone or of ligature of the carotid. Even when under these circumstances they do occur, this may indicate, as I have said above, the existence of anomalies in the circulation. On the other hand, the temporary hemi-pareses and paralyses that follow ligature of the carotid, are evidently due to anæmia of the cerebrum itself, as is further shown by the fact of their disappearance after the establishment of a collateral circulation.

Persistent paralyses are, probably, always due to anatomical changes, usually softening of the cerebral substance, such as are distinguishable after death.

Clinical History and Prognosis.

It is impossible to lay down general rules governing the prognosis and course of cerebral anæmia ; they differ for every case.

Simple faintness from mental influences runs almost invariably a favorable course, the patient soon returning to a normal state ; yet now and then a fatal case is met with.

As regards recurrence, the cases obey no law ; the attacks may occur but once in a lifetime, or, as in the case of an hysterical person described by P. Frank, several times in a single day.

Greater reason for anxiety attaches to syncope, such as sometimes attacks convalescents when they raise themselves upright for the first time ; it lasts longer than simple faintness ; it is not an unheard of thing that it should result in death.

Still more true is this of cerebral anæmia from profuse hemorrhage ; although occasional losses of blood are less to be feared than repeated bleedings.

If it is really true that the symptoms of shock are to be laid to the score of cerebral anæmia, produced in the manner indicated above, then this would constitute a form of anæmia which, as is well known, not rarely ends fatally.

The prognosis in a case of hydrencephaloid must be based upon a consideration of the treatment which has been adopted. If the child was not excessively feeble to start with, it will generally recover under judicious care. Dangerous as the symptoms appear, they will usually pass away at last. This is perhaps still more true of the delirium of anæmia. It happens only exceptionally that, under proper treatment, these disturbances of the mind are prolonged ; still more rarely are they incurable. In case of anæmia due to heart disease, or to gross changes within the cranium, the progress of the secondary affection will be of course determined by that of the primary disease. and naturally the prognosis will generally be unfavorable.

It need hardly be said that the prognosis may also be determined in part by the severity of the symptoms: thus it will be favorable—that is, so far as the anæmia itself is concerned—without regard to underlying disorders, if these are of the lighter sort, consisting in dizziness, tinnitus aurium, etc. less so if convulsions or deep and prolonged coma be present.

The state of the pupils demands consideration, as giving, according to the clinical and experimental experience of Kussmaul, material help in the formation of the prognosis: at the outset of an acute attack of anæmia they become temporarily contracted; as it goes on they dilate, and finally, with the gradual return of the circulation to its normal state, they resume their usual size.

Treatment.

General rules are applicable here only to a limited extent, each case requiring a treatment of its own. Among them is to be mentioned the importance of giving the body such a position as will favor as much as possible the flow of arterial blood to the brain, that, namely, in a horizontal plane. The enforcement of a certain position is, to be sure, only an accessory in the treatment, and sometimes, as in the case of delirious patients, it is not to be carried out; but its importance is shown by the well-known fact that persons who undertake to raise themselves up suddenly from the horizontal position, after having been copiously bled, will often faint away (hence the old rule, that venesection should be done with the patient in a sitting posture). In many cases—in those of the kind just mentioned, as well as in the syncope of convalescents—giving the head a relatively low position in this way, is all that is needful; in any event, it does no harm.

The principles of treatment must vary according to whether the anæmia is limited to the brain or affects the entire body; also according to whether it is acute or chronic.

In the common form of syncope, such as is excited by emotional impressions, or occurs in convalescents, or in consequence of temporary hyperæmia in other parts of the body, the symptoms usually disappear of themselves under the influence of the horizontal posture.

If the symptoms are more severe and persistent, smart irritation of the skin is useful, as produced by sprinkling the face with cold water; this has a stimulant reflex effect upon the respiration as well as on the action of the heart. The most powerful of these irritations is produced by the metallic electric brush; less useful are the mustard applications, since they are superfluous in the lighter cases, inefficient in the more severe.

Of greater value are the agents which act on the nerves of the mucous membranes, such as the preparations of ammonia and other substances which irritate strongly the trigeminus or olfactorius, or irritating enemata containing salt and vinegar. If the pulse is much enfeebled, direct stimulants for the heart may be necessary, such as coffee, alcohol, or, better, the quicker-acting champagne, or ether. Finally, in case of unusually severe, prolonged anæmia from loss of blood, transfusion should be tried as a last resort.

The therapeutics of hydrencephaloid have been studied especially by the English practitioners. It is scarcely necessary at the present day to warn the readers against the danger of confounding this affection with cerebral hyperæmia or meningitis, and against the use of antiphlogistic treatment. The influences which produce the anæmia, the cholera-infantum, etc., must be removed, and at the same time general tonic and stimulating agents employed.

Of these are especially to be mentioned, wine, in doses proportioned to the age of the patient, soup, and milk; the early use of musk is to be recommended.

One of the most important means of treatment is, however, careful and thorough warming of the surface of the body. The cold douche for the head, which is sometimes employed, is, if not actually injurious, certainly without effect upon the general course of the affection; at the best it provokes a remission of the sopor, but does not influence the fundamental trouble. If the opposite group of symptoms, the delirium of inanition, be present, the most important step is again to remove the general anæmia by good nourishment; and if the action of the heart is depressed, wine is here also indicated. If there is not as yet actual delirium, but only an abnormal degree of mental excitability, and restlessness, cutaneous irritants, even though but slightly painful, such as mustard and blisters, are to be avoided, as indeed Andral observed, because they tend to increase the irritation of the central nervous system.

Against conditions of extreme excitement the opiates are the sovereign remedy, and act often surprisingly well; even after a single injection of morphia, if only enough to induce sleep, the patients often awake with restored sensorium. Since the opiates are known to act with disproportionate intensity upon enfeebled subjects, the dose must be carefully chosen (a fourteenth or a tenth of a grain of morphia; at the most, a fourth of a grain). I have frequently found chloral also to work well in these cases.

As we have here to do only with anæmia of the brain as such, and with the treatment only of the conditions which are directly dependent upon that affection, we need not discuss the therapeutics of the primary underlying disorders which may exist.

HYPERÆMIA OF THE BRAIN.

Compare the standard general literature and the works represented in connection with the subject last treated of; further: *J. Hope*, Inflammation of the brain and its membranes.—*Landois*, Ctrlbl. f. d. medecinischen Wissensch. 1867.—*L. Hermann* and *Th. Escher*, Ueb. die Krämpfe bei Circulationsstörungen im Gehirn. Pflüger's Arch. III. Bd.

Historical Sketch.

The single steps in the history of the doctrine of hyperæmia of the brain are much harder to follow than in that of anæmia. From the earliest times it has held a recognized place among physicians as a distinct pathological condition; Galen indeed affirmed the existence of apoplexy of congestive origin.

Again, in the works of Boerhaave (l. c.) is found a much clearer and more thorough discussion of the subject than in those of almost any of the later writers of the last century.

Altogether the best *clinical* description of the affection to be found among the older writers is that of Andral, abounding as it does in acute, independent observations.

In the teachings of Kellie we find the subject of the practical relations of cerebral congestion passed over in comparative silence; to go into it at length seemed unnecessary. The theoretical points at stake have been studied much less than those involved in cerebral anæmia; early experimental investigations, for instance, are wanting, except for those of Kellie himself, and it is only very recently that any such have been undertaken.

In recent times there has been a reaction from the opinion of former periods, when the diagnosis of cerebral hyperæmia was made to cover every variety of symptom, so complete that from some quarters much doubt has been thrown upon the clinical importance and even the very possibility of occurrence of this affection.

Although acknowledging fully that the evidence brought forward to sustain the opposite doctrine is open to much criticism, yet it would be certainly going too far to turn, in critical zeal, the cold shoulder upon cerebral hyperæmia altogether. In our opinion it is a disease of decided clinical importance.

Etiology.

A great variety of widely different causes have long been considered as important in the production of cerebral hyperæmia.

As in the etiology of anæmia, so also in that of the reverse condition, a division line is to be drawn between those influences which cause hyperæmia limited to the cranial contents, and those which induce a similar condition in other parts of the body as well. Here also, as there, we find causes which are temporarily, others which are permanently active; further, only such as induce diffused, and such as induce local congestion within the cranium. Furthermore, it is customary to distinguish between the causal influences according as they give rise to an active arterial hyperæmia or to a passive venous stasis. From this point of view, the *hyperæmia of fluxion*, i. e., *active congestion*, is to be separated from the *hyperæmia of stasis*, á distinction which is also of clinical importance. Finally, those causes are to be considered which do not act directly and immediately, but which tend to excite a morbid predisposition.

Although many of the conditions, shortly to be considered in detail, may unquestionably give rise to cerebral hyperæmia, still the question whether definite existing symptoms are actually dependent thereon is often very difficult to answer; in short, there is as much room for hypothesis here as in the case of cerebral anæmia, for experimental evidence sufficient to furnish a sure guide to diagnosis is wanting. Let us consider, first, certain conditions which are generally admitted to be predisposing causes.

A certain temperament, the so-called "*habitus apoplecticus*," is said to favor the occurrence of active congestion. We shall discuss this supposed connection later in speaking of hemorrhage, and will only venture the statement here that it is neither so frequently nor so unmistakably met with as is often supposed—if indeed it exists at all. Experience seems rather to justify the conclusion that very fat and very muscular persons show a greater predisposition to cerebral hyperæmia than persons of normal nutrition: yet with the difference that in the fleshy class the tendency is rather to stasis, in consequence of the disturbances of respiration so common among them—with the muscular, rather to active fluxion, in consequence of the unusually great activity for which the heart is called upon.

Equally uncertain is the significance of the so-called *plethora universalis*. Even admitting the fact that persons of the well-known appearance indicated by this term are more likely to suffer from dizziness, headache, tinnitus aurium, and the like, than persons in health, yet we cannot but acknowledge, with Andral, that it can by no means be regarded as proved that these symptoms are to be laid to the score of cerebral hyperæmia. As he remarks, such phenomena might well be due to a qualitative as well as to a quantitative change in the blood circulating through the brain (consisting, as he believed, in an increase of the red corpuscles). To come to a definite opinion in the matter seems to us as yet impossible.

The character of the diet used is more or less important in this connection, but plainly only in so far as it affects the general nutrition of the body, helping, in certain cases, to an abnormal production of fat; or, in others—together with the occupation of the patient—to an unusual development of the muscular system.

It is certainly an error to attribute to aged persons a disproportionate tendency to cerebral hyperæmia; the mistake has plainly arisen from the confounding of this condition with hemorrhage, the frequency of which in persons of advanced age is due to a totally different set of causes. We have, to be sure, no statistical evidence to offer, yet daily experience teaches that the affection is as common in (comparative) youth as it is in old age; at the same time the relative frequency of the special varieties of the disease (classified according to their etiology) may vary at the different ages—the vaso-motor fluxions, for example, being commoner in younger, the venous stases in older persons.

That hereditary predispositions exist, still requires demonstration. The assumption appears in so far justifiable that the habitus apoplecticus and the plethora universalis may be, to a certain extent, hereditary. Of still more uncertain significance than the general causes which have just been considered are certain others, to which greater or less weight has been attached, such as the influence of the seasons. The important element in this influence is probably the action of the temperature—a point to which we shall refer again below. At the moment we need only recall the apparently surprising fact that, according to the coincident, though not extensive figures of Andral and Falret,[1] cases of cerebral congestion are more common in Holland and in Turin during winter than during summer. The statistics given by Hammond, from his own experience in 622 cases, tend to favor the same conclusion: between September and November there were 110 cases; between March and May, 131; between June and August, 179; between December and February, 202. As to the importance of the part, if any, played by the different winds, we have no reliable evidence.

The influences which are particularly active in producing cerebral hyperæmia might be grouped according to whether they act temporarily or permanently, whether inside or outside of the cranium. From a clinical stand-point, however, the really important distinction is to be made between those which excite active congestion (fluxion) and those which excite passive congestion (stasis).

Let us first take up the *causes of active hyperæmia.*

Of these, the chief consists in *increased activity of the heart.* It is not necessary for this that the heart should be hypertrophied; let a perfectly normal heart be, for any reason (emotional excitement, etc.), urged to abnormal activity, and cerebral congestion, overfilling of the arteries and capillaries, may follow. It is often believed that this result occurs *more* readily in case of hypertrophy; and this is said to be especially true of the so-called idiopathic hypertrophies.

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[1] Quoted by Andral.

We have not been able to satisfy ourselves absolutely with regard to this point; but it is easier to credit this than to believe that the secondary hypertrophies, such as accompany valvular diseases, should give rise to cerebral congestions. Under these latter circumstances the extra strength of the hypertrophied ventricle is sufficiently occupied in overcoming the abnormal resistance which is presented to it. On the other hand, the case is evidently somewhat different with hypertrophies following contraction of the kidney, which might certainly be supposed to act as a predisposing cause of temporary active congestions.

The clinical phenomena impel us to seek the cause of active congestion, in other cases, in affections of the *vaso-motor* system.

It sometimes happens—occasionally with men, oftener with plethoric and nervous women—that dizziness, headache, etc., occur in connection with attacks of flitting heat and redness, sometimes passing almost into cyanosis, in the region of distribution of the carotid, at times without emotional excitement or other demonstrable cause.

This condition is often entirely unattended, as I have observed, by palpitation or changes in the pulse.

To explain these cases, one is obliged either to assume that certain nerves having the functions of directly dilating the blood-vessels, whose existence has been, to say the least, shown to be probable, by the experiments of Schiff and Goltz, are excited to action, or else that a reflex paralysis of the vaso-motor nerves has taken place, an occurrence which has been experimentally demonstrated to be possible. Whether any affection of the depressor nerves ever helps to produce the effects in question cannot be stated with certainty, though at the same time we do not consider that too much weight ought to be attached to the negative results of the few experiments of Jolly, who failed to observe increased vascularity of the brain after irritation of the depressor.

We agree entirely with Basch,[1] in his opinion that the so-called vertigo e stomacho læso is not to be laid invariably to cerebral anæmia, but that some cases, if we may be guided by the clinical phenomena, are probably due, to speak in general terms, to reflex cerebral congestion. Bouchut[2] considers that a group of symptoms completely simulating meningitis may arise from a congestive affection of the brain.

A number of writers, especially Möllendorf,[3] Eulenburg and Guttmann,[4] O. Berger,[5] have tried to show, also, that one form of migraine is caused by a unilateral, neuro-paralytic, cerebral hyperæmia. There is certainly much to be said in favor of this view, although absolute proof is wanting.

That *medicaments* may induce an intracranial congestion, though oftener asserted than proved, is yet, for certain drugs, beyond a doubt.

At the same time, the symptoms due to hyperæmia, under these circumstances, are usually entirely overshadowed by the effects of the direct action of these poisonous agents upon the cerebral tissues.

In opposition to the usual assumption, that various narcotics, especially opium, belladonna, and hyoscyamus, excite cerebral congestion, we feel bound to state that this is by no means certain. On the other hand, as regards *alcohol*, direct proof of such an action has been furnished by the experiments of E. A. O. Neumann, indirect proof also by those of Kremiansky.[6] A still more marked hyperæmia than that induced by alcohol seems to follow the inhalation of *nitrite of amyl* (Richardson, Brunton, and others). In this case it is due, however, not to active fluxion, but to paralysis of the vascular nerves.

Prolonged and intense mental activity is also usually regarded as a cause of cerebral hyperæmia. Although we are not in a position to deny this, in face of the direct statements of such an observer as Andral, and indeed in face of the results of post-mortem observations (Niemeyer), still it appears to us highly improbable that the symptoms which attend this excessive mental action are exclusively, or even mainly, due to cerebral hyperæmia. Is it not far more probable that the unusual activity of the ganglion-cells themselves ought properly to be held responsible for these symptoms, though the manner of its action is as yet unknown?

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[1] Ueb. den Magenschwindel u. verwandte Zustände. Wien. med. Presse, 1873.

[2] Des névroses congest. de l'encéphale. Gaz. des Hôp. 1869.

[3] Ueb. Hemicranie. Virch's Arch. 41. Bd.

[4] Die Pathol. d. Sympathicus. Berlin, 1873.

[5] Zur Pathogenese d. Hemicranie. Virch's Arch. 59. Bd.

[6] Virch's Arch. 1868.

The assumption, that the direct impingement of the sun's rays upon the head may excite an active congestion, is universally accepted as fact. That this assumption is probably correct, is true enough, but it is by no means proved. When the cerebral symptoms, apparently due to the heat of the sun, occur, as is the rule, in the course of bodily exertions, the increased activity of the heart excited by the latter is surely to be taken into account as a possibly important factor.

Again, as for the cerebral congestion from which the well-known group of symptoms characterized as "*sun-stroke*" or "*heat-stroke*," were formerly assumed without question to arise, it has been placed beyond doubt that the direct influence of the sun has nothing whatever to do with its production, and that the hyperæmia discovered after death is venous in character, and a secondary phenomenon, immediately dependent upon a diminished power of activity of the heart. Both clinical and experimental observations (Obernier[1]) have shown that the cause and nature of sun-stroke are to be sought in the abnormal increase in the temperature of the body, which exerts its effect in a manner not to be described here.

Following the indication furnished by this discovery of the nature of sunstroke, we are at liberty to conclude, as has already been shown by Liebermeister,[2] that the cerebral symptoms in *fever* are only to a limited degree, if at all, dependent upon cerebral hyperæmia. It was formerly assumed as an axiom that in high fever, as for example in typhoid, scarlatina, pneumonia, and the like, active cerebral congestion occurred as a matter of course; no proof of this was ever given, but it was inferred from the turgor faciei, and because no other explanation of the cerebral symptoms could be suggested. Now, that we know, through the observations of Heidenhain and others, that the cutaneous circulation in fever is of a different character from that of the interior of the body, and that the turgescence of the face does not indicate a corresponding condition within the brain; and now that we are able, with more than probable correctness, to refer the symptoms in question to the abnormally elevated temperature,

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[1] Der Hitzschlag. Bonn, 1867.

[2] Ueb die Wirkungen der febrilen Temperatursteigerung. Deutsch. Arch. f. kl. Med. I. Bd.

—we need not, *ad hoc*, assume a cerebral congestion, and may fairly regard it as at least unproved that fever gives rise to such a condition.

Finally, much light is thrown upon the etiology of congestive hyperæmia by a study of those cases in which blood is determined to the head in consequence of partial or complete shutting off of the arterial blood current from other parts of the body—the so-called *hyperæmia* from *collateral fluxion.* We do not refer here to that form of collateral fluxion which occurs in connection with circumscribed diseases of the brain, such as thrombosis and embolism, hemorrhage, abscess and tumors, which will be treated of in another place. But there are other morbid affections which are often followed by hyperæmia within, and especially erysipelas capitis.

Those cases are also of great clinical importance where, from causes at present not understood, the blood-supply of various parts of the body, now one, now another, and among them the brain, becomes temporarily increased ; persons subject to these attacks are sometimes plethoric, sometimes not. Well-marked typical cases of the kind, such as the one described by Andral (Clin. Méd., V. éd., p. 269), are met with, to be sure, but seldom ; but, on the other hand, we often see patients who suffer for a while with frequent attacks of nose-bleed, then complain of dyspnœa, with or without palpitation, after the subsidence of which the symptoms of active hyperæmia of the brain make their appearance. At other times the symptoms of cerebral congestion attend the *cessation of normal or pathological secretions*, of regular losses of blood : thus it may occur on the stoppage of hemorrhage from piles ; sometimes from suppression of the menstruation, or in the climacteric period. The symptoms in these cases, almost without exception, are of the lightest kind.

The anatomical conditions are more distinct in those cases where there has been a demonstrable occlusion of certain large arterial districts : a pregnant example of this is the hyperæmia on one side of the brain which follows ligature of the opposite carotid ; sometimes it happens that the symptoms point to an affection on the same side with the ligature.

This is the place to speak also of another influence which gives rise to cerebral symptoms believed to be due to a collateral hyperæmia, viz., *that of severe cold.* We would not deny that this may effect a repulsion of the blood from the surface of the body towards the internal organs. At the same time, it is not known with certainty that the blood pressure is thereby made to rise; there is rather reason to think that the power of the heart is diminished, which would be likely to cause venous stasis.

When, however, the results of experiments are taken into account, especially those of Horvarth,[1] who found, as a consequence of the cooling down the bodies of animals, that the temperature of the blood in the carotids diminished as well as that of the rectum, it cannot but be believed that hyperæmia (whether venous or arterial) plays but an unimportant part in exciting the cerebral symptoms which follow exposure to severe cold. On the other hand, the lowness of temperature of the blood circulating in the brain must, we think, be regarded as the chief factor in their production.

Much clearer, and, as a rule, less open to question are the etiological relations of the *hyperæmia by stasis.* Venous stasis in the region supplied by the vena cava sup., including of course the brain, must always occur when any obstacle exists to the free passage of blood from the right ventricle, whether from a pre-existing overfilling of the pulmonary arteries, or from an actual diminution in capacity of the lesser arterial system—the intracranial stasis forming one part only of the general venous congestion.

At the same time experience has shown that such a stasis occurs much more readily in the vascular system of the inferior, than in that of the superior cava—the former sometimes giving rise to well-marked symptoms before the latter has betrayed itself in any way.

Among the special morbid processes which cause the condition just mentioned, the most common are certain *diseases of the lungs*, such as the emphysema of Laënnec, large pleuritic effusions, extensive pneumonias.

It scarcely ever happens, unless through accidental complications, that venous hyperæmia of the brain accompanies phthisis, no matter how great the mass of the destroyed lung—and this simply because the disease, from its long duration, has excited a condition of general anæmia.

Certain affections of the heart, such as stenosis of the left venous orifice and insufficiency of its valves, act in the same manner as the pulmonary diseases mentioned.

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[1] Various communications in the Ctrlbl. f. d. med. Wissensch. Jahrg. 1870–1873.

In all these cases the stasis comes on—as need not be demonstrated at length—only in case the compensatory hypertrophy of the right ventricle has not yet become sufficient to overcome it, or has ceased to be so. Thus in the last stages of aortic disease, or in case of large pericardial exudation, venous hyperæmia of the brain may occur, though, to be sure, not in the typical form, but conjoined with anæmia. Tricuspid insufficiency may give rise to such stases even more readily than the valvular lesions already mentioned.

It is plain that venous hyperæmia of the brain will always attend any interference with *the passage of blood through the jugular vein.* If the vessel of one side only is closed, its work may, after a time, be done by that of the other side. We need not speak in detail of all the possible anatomical conditions, tumors in the cervical regions, etc., by which these effects may be produced. One class of cases alone claims our special attention: those of *suffocation.*

From the earliest times marked cerebral hyperæmia had been held to follow throttling, compression of the jugular veins, and of the trachea. Ackermann was led, in consequence of experiments of his own, to oppose this view. He was only able to discover a temporary cyanosis under these conditions, and that only when the strangulation was not at once complete, or when the respiration alone was cut off; this was followed invariably by anæmia of the brain. Jolly confirmed the latter part of the observation, but found that the anæmia only made its appearance at death, while hyperæmia was present so long as life endured. Further experiments are needed to decide this question. If the justice of the earlier assumption is maintained, then not only the closure of both jugulars, but also *stenosis of the larynx* must be reckoned among the causes of venous cerebral hyperæmia. This must, of course, happen much oftener in the acute forms of this condition (croup, œdema of the glottis, presence of foreign bodies) than in the chronic. The influence of thrombosis of the sinuses, which may also constitute the cause of venous stasis, will be treated of in a separate chapter.

The foregoing are, however, not the only causes of venous hyperæmia of the brain. There are still a number of influences which are rather transitory in their action, such as all forced expiratory efforts made with closed glottis. The venous stasis in the parts supplied by the superior cava, which attends attacks of coughing, is familiar to all, occurring especially when the coughing comes on in severe paroxysms, as in tussis convulsiva, in emphysema, etc.

Violent screaming, and long-continued blowing of wind-instruments has a similar effect; further, powerful compression of the contents of the abdomen, such as occurs in efforts to empty a constipated bowel, or during child-birth. The cerebral hyperæmia attending the second stage of an epileptic attack also deserves mention here. Whether the dilatation of the stomach by food can have any effect worth naming on the intracranial circulation is not yet definitely settled; while some observers (for example, Durham) judge from the symptoms that anæmia is present, others from the same evidence (for other indications are wanting) that there is hyperæmia; we share in the latter view. Finally, the influence of the position of the body is to be mentioned—every position, in which the head is lower than normal, exciting naturally hyperæmia of the brain.

Pathological Anatomy.

The symptoms during life and the post-mortem appearances do not always stand in direct proportion to each other. Slight, transitory hyperæmias of the brain, especially those of the active, arterial kind, may disappear without leaving a trace of their presence, just as often happens in other parts of the body. Further, the same may be true of the venous stases as well as of those due to suffocation, as has already been pointed out. Ackermann and Jolly, though disagreeing with each other in certain points, were both led by their experiments to the same conclusion, that the brain after suffocation appears after death anæmic; and the former declares that, even when an appearance of congestion is found, this is due partly to post-mortem hypostasis, partly to the greater tendency to diffusion possessed by the blood after suffocation. On the other hand, as numberless observers have stated, large collections of blood are often found in the dependent parts of the skull cavity—which in case of dorsal decubitus will be the occipital fossa—generally enclosed in the veins and sinuses, which has made its way there under the influence of gravity, either post-mortem or even during the last few days of life, in the opinion of some of the older writers, and latterly of Laborde,[1] the same thing being held to occur also with the lungs.

Hyperæmias of this kind are fairly to be considered, from their position, as post-mortem phenomena. Again, attention may be called to the fact, that a normal, moderate fullness of the veins is often regarded by novices as a pathological appearance; yet, in speaking of cerebral anæmia, we mentioned that even here the veins appear tolerably well filled.

Even in the severer cases of hyperæmia, in case it has not lasted too long and become fixed, the venous form is much more easily recognizable after death than the arterial. Even in the tissues over the cranium the amount of blood is often found to be abnormally great.

When the calvarium is removed, drops of extravasated blood from ruptured vessels are observed on the dura. The veins of the dura stand out flushed with blood, still more those of the pia; the choroid plexuses and the sinuses are similarly crowded with blood.

In the intenser forms of hyperæmia, in persons of middle age or below, the brain is found swollen, and pressed against the dura, and the gyri flattened.

The brain-tissue, especially the gray substance, is altered, of a darker color than normal, and drops of blood make their appearance in greater or less abundance on the cut surface; this occurs especially in the white substance, although the general darkening of color is there but rarely seen. The perivascular spaces disappear (according to Golgi).

Much more pronounced are the changes which frequently recurring active hyperæmias or chronic stases leave behind them.

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[1] Le ramollissement et la congestion du cerveau. Paris, 1866.

These changes affect, in the first place, the *vessels themselves.* After venous stasis the veins are found enlarged and tortuous, especially those on the surface of the brain and in the pia. After arterial congestion the small arteries also remain dilated. In both cases even permanent small aneurysmal dilatations may be formed, especially on the capillary vessels, as Ecker,[1] Schroeder van der Kolk,[2] Hasse, Laborde, and others have shown.

Strongly marked hyperæmia may also cause small *capillary hemorrhages*, whose position is indicated post-mortem by minute yellow or yellowish-red points. Furthermore, Charlton Bastian[3] calls attention to diffused collections of amorphous blood-pigment, which lie along the vessels, in the perivascular spaces, and in his opinion point with certainty to the previous existence of hyperæmias of long standing. It has not yet been discovered just how the blood-pigment gets outside the vessels in these cases.

Since it is well known that the abnormal fullness of the vessels is associated with an elevated blood-pressure, it is entirely comprehensible that a *transudation* of the blood-serum should also occur. In this way œdema of the pia, swelling of the choroid plexuses, and increase of the ventricular fluid is produced. The question whether the hyperæmia can go over into inflammation, which was formerly answered categorically in the affirmative, later called again more and more in question, need not be discussed at length, in view of our present acquaintance with the theory of inflammation. As a further consequence of hyperæmia, cerebral atrophy may be developed, probably in consequence of the capillary anæmia and accompanying disturbance of nutrition to which the pressure of the fluid transuded (according to the laws of the intra-cerebral circulation above described) under the elevated vascular tension, gives rise.

Since the observations of Durand-Fardel, the condition designated as état criblé has also been usually regarded as a consequence of long-standing hyperæmia. This sieve-like appearance of the brain is characterized, as is well known, by the presence on the cut surface of numberless round or oval openings, sometimes as large as a pin's head. These holes were formerly referred entirely to enlargement of the small vessels; but Bizzozero, Golgi, Bastian, and others have proved that they depend still oftener upon dilatation of the perivascular lymph-sheaths. These dilatations are met with principally in the white substance of the hemispheres, in the corpora striata, and the optic thalami.

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[1] Diss. de cerebri et med. spin. system. vasor. capill. etc. Trajecti ad Rhenum, 1853.

[2] Over het. . . .verlengde ruggemerg. etc.

[3] Congestion of the Brain, in Reynolds's System of Medicine.

They are oftenest found at the autopsies of elderly persons and of those who have suffered from stasis of long duration.

The question as to the occurrence of *partial hyperæmias* still remains to be discussed.

The clinical symptoms seem at times to require the assumption of this pathological condition. Anatomically its existence may at times be demonstrated—as, for example, in the neighborhood of new-growths, localized inflammations, and the like, and, further, where local stases follow in the train of local obliteration of vessels.

It is, however, quite another question whether active congestions can limit themselves to one side or circumscribed regions of the brain.

It has never been anatomically proved to our satisfaction that this is possible, nor have we been able to convince ourselves of the fact.

In the cases reported by Calmeil for instance (l. c., Tom. I. 8th and 9th Obs.), where the vascular injection on one side was greater than that on the other, inasmuch as no symptoms, such as might have been due to this condition, had been observed during life, it may fairly be referred to a post-mortem hypostasis. In Andral's cases, on the other hand, where well-marked unilateral symptoms had been present (Clin. Méd., T. V., 2d and 3d Obs., pp. 221 and 227), no difference was found as regards the intensity of the congestion between the two sides of the brain.

Nevertheless, reasoning from analogy, the possibility of such an occurrence seems to us entirely admissible. Since it is possible that circumscribed regions of the face may redden under emotional excitement, why should not the same be true of the brain? There is at least no convincing argument to be adduced against this view. We find, moreover, that it was expressed by Rostan,[1] and still more emphatically by Graves.

Experimental Investigations

have been made unfortunately only in very limited number, and they have reference, of necessity, only to venous hyperæmia following an interruption to the return of blood from the brain.

Landois found, with rabbits, that under these circumstances a diminution in the rapidity, sometimes even complete cessation, of the heart's action (from irritation of the vagus-centre), occurs. When the slowing has reached its maximum, general convulsions come on, as in consequence of cerebral anæmia; synchronously with this appear the symptoms of irritation of the vaso-motor centre (general contraction of blood-vessels and tense fullness of the heart). Hermann and Escher also succeeded with cats, though they failed to do so with rabbits, in exciting epileptiform convulsions by completely checking the return of venous blood from the brain. These convulsions were preceded by dyspnœa, and were to be distinguished from those of anæmia only by their somewhat tardier beginning.

Jolly has further shown that, with dogs, even the simple compression of the jugular veins causes an increase of the intracranial pressure.

We have already called attention to the experiments of Ackermann upon the effects of suffocation.

Symptomatology.

Although the occurrence of cerebral hyperæmia has long been assumed, and various symptoms have been referred to it, yet it is, for several reasons, much more difficult to describe the latter systematically than it is those which are indicative of cerebral anæmia, with which our acquaintance is so much briefer. First of all, we have no sufficient experimental evidence to enable us to decide with certainty just what symptoms are really characteristic of hyperæmia; with regard to the passive form, a few experiments are recorded, as we have seen; but as regards the active, absolutely none.

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[1] *Léon Rostan*, **Recherches sur le Ramollissement du Cerveau, etc. Paris, 1823.**

Next to be mentioned is the want of proportion, of which we have spoken, between the clinical and the post-mortem phenomena.

Finally, cerebral symptoms have often been referred to hyperæmia, which have had nothing whatever to do with it, to the complication of its proper symptomatology. Thus, Andral has described not less than eight forms of the disease, certain of which, as the analysis of his cases shows, are surely not pure instances of cerebral hyperæmia—if, indeed, they deserve the name at all.

To base the assumption of different pathological conditions upon differences between single, relatively prominent symptoms, is, in our opinion, an entirely arbitrary method of classification.

The only allowable distinction seems to us to be the clinical one between *light* and *severe* cases, the former being by far the more common of the two.

Sketch of the lighter forms.—The patients, who may be young or old, of plethoric appearance or not, complain of quite severe dizziness, of pain in the head, and a sense of confusion.

These symptoms may recur off and on, lasting from half an hour to an hour, or may persist for one or more days. During this time the patients are often excited, sensitive to light and noise; their temperament seems changed; they appear peevish and irritable; the intellectual functions become impaired; they are forgetful and unable to work. Hammond lays great stress upon the existence of sleeplessness, as one of the earliest and most important symptoms.

The face is sometimes reddened or even livid; the arteries of neck and head beat forcibly, although there is not necessarily palpitation of the heart; a sense of flying heat shoots over head and neck. Sometimes nausea and a tendency to vomit comes on, but very rarely actual vomiting. Such is the light form of active congestion.

If the cerebral hyperæmia is due to venous stasis, for example, in consequence of heart disease, the patients suffer, as above, from dizziness, tinnitus aurium, confusion of ideas, and a dull sense of oppression in the head, and, as before, the face is reddened (of course in consequence of the abnormal fullness of the veins), but the mental excitement and excitability are wanting; and instead, there is a certain mental torpor with a tendency to sleep; the beating of the arteries is also absent, and all these symptoms are more continuous than those mentioned above, and not paroxysmal.

That these lighter groups of symptoms stand in mediate or immediate connection with cerebral hyperæmia is beyond question. It is, on the other hand, not so clear that all the other symptoms whose presence is said to characterize the *severer* forms of cerebral hyperæmia are really always dependent upon this alone; such are: delirium, attended with attacks of actual mania, coma, convulsions, paralysis, fever, and finally all the signs of meningitis.

We think it wiser, therefore, not to treat of these symptoms as representing different types of the disease (hyperæmia apoplectica, epileptica, maniaca), but to discuss them singly on their own merits.

A comparison of the symptoms of cerebral hyperæmia with those of cerebral anæmia discovers points of likeness, which are so striking that they explain how it happened that the two conditions were formerly so often confounded one with another; in both we meet with derangements of the sensorium, the organs of special sense, the sensitive and motor nerves.

Disturbances of the sensorium.—Even the earlier writers (Hope, for example) lay stress upon the fact that of the various mental disturbances occurring in hyperæmia, those with the character of exaltation belong rather to the active, those of the nature of depression, on the other hand, rather to the passive form of the disease.

For the majority of cases this is true enough, though the rule is not universal. As regards symptoms of *mental irritation*, the patients, in the lighter forms, are excitable, fretful, restless; during the continuance of the hyperæmia sleep is insufficient, and disturbed by dreams; yet the faculty of speaking and acting intelligently is not lost. With the disappearance of the anæmia the normal state returns.

That a certain garrulousness, a restlessness of such a kind that the patients talk and behave unnaturally, get out of bed at night and move about their effects aimlessly, and the like—a condition described by Durand-Fardel as occurring among the aged—is to be referred to cerebral hyperæmia, seems to us a matter open to great doubt, certainly as not proved.

We have already stated it as our opinion, with regard to the matter of febrile excitement (delirium, etc.), that its connection with cerebral hyperæmia is difficult to demonstrate. On the other hand, there are certain conditions of mental exaltation whose occurrence is hardly to be explained otherwise than by supposing them to be due to cerebral hyperæmia. The patients are delirious, excitable, and may even fall into *attacks like those of mania.* The following case, observed by me through a considerable period, is a good example of the kind:

A shoemaker, fifty-seven years of age, born of healthy parents, had been subject since his fourteenth year to periodic attacks of slight excitement. Up to his twentieth year he suffered often from "rush of blood to the head," with a sense of fullness, heat and pain in the head; a nose-bleed always brought him relief. From his twentieth year to the present time he has been seized from time to time with peculiar paroxysms of excitement, recurring at first every four, then every six or eight weeks, latterly four or five times yearly. The nature of these attacks is as follows: The patient is seized with intense palpitation of the heart, accompanied by a sense of oppression, a feeling of heat rises to the head, specks appear before the eyes, tinnitus aurium comes on, yet the force of the heart's action is not increased, and there is no dizziness. The face becomes somewhat reddened, but not excessively so.

The patient then falls into a curious state of bodily and mental excitement and irritability, wanders restlessly around, not only in his chamber, but through the entire village, refuses to work, becomes excessively unruly and pugnacious, cries out and rages about, but withal has no real delusions. This state persists for a few hours or days. The drinking of large quantities of water procures the patient a degree of ease; but it is especially striking how quickly the symptoms fade away after venesection. The physician in charge has therefore been in the habit of drawing regularly a small quantity of blood, varying from 150 to 300 grm., and has had to repeat this treatment with nearly every attack for the past thirty-four years. The patient himself would not suffer it to be omitted, even if the physician desired it, but would insist until the venesection was performed, and the customary relief obtained. Except for the fact that the patient makes the impression of a person with rather limited mental calibre, there is nothing abnormal to be observed about him, and, especially, not a trace of mental excitement to be seen during the intervals between the attacks.

In other cases the patients have actual delirium and well-marked delusions. On the other hand, we would most emphatically warn against the fashion of referring *permanent* states of mental excitement or depression of every kind to anæmia and hyperæmia of the brain.

The habit of doing this is greatly in vogue at the present day. That changes in the circulation may exert an influence upon the mental functions, has been placed beyond question, and has been uniformly affirmed by us, as in these pages; nevertheless no proof has yet been given that, by themselves, such changes can cause permanent symptoms of mental disease; and if anatomical alterations are now and then discovered after death, it can by no means be affirmed that they are primary and not secondary in nature. Under certain conditions, *states of mental depression* are also to be met with as symptoms of cerebral hyperæmia—the patients appearing dull and listless, and inclined to rest and sleep. The mind is confused, and distinct thoughts and perceptions are impossible. This is seen, in particular, in cases of passive stasis, due to certain diseases of the heart, and the like; and under these circumstances this condition is likely to persist for some time. It may be produced transitorily, at will, by the energetic performance of such movements as hinder the return of blood from the brain.

A special form of cerebral hyperæmia has been recognized under the name "*apoplectic*." Boerhaave speaks of an apoplexia e plethora; and in France the same condition is known as coup de sang.

After lesser symptoms, pointing to a disturbance of the sensorium and the nerves of special sense have gone before, sometimes indeed suddenly and without premonition, the patient falls into a state of coma. After a period, varying from a few minutes to half an hour or twenty-four hours, he awakes, is confused, his speech is slightly labored, but otherwise he shows no sign of paralysis, and he returns gradually to a normal state. The consciousness is not always totally lost; sometimes the patient answers loud calling by signs, and even attempts to answer in words, though his speech is confused and labored. In other cases, such patients, though they may never have shown symptoms of disturbance of the nervous system, do not wake up, but die in the coma, and the autopsy reveals nothing but pronounced hyperæmia. Cases of this latter kind (known as apoplexia serosa among the older physicians) are not common, but there can be no doubt that they occur; Andral, for instance, described a typical instance of the sort (l. c., p. 218), and Hammond speaks of two. We should be all the more careful for this reason about making the diagnosis of "*congestive apoplexy*" in case of the occurrence of the transitory symptoms above described.

We would not deny that typical forms of active hyperæmia *may* give rise to these transitory symptoms, but we cannot but doubt whether the occurrence is so very common as has been believed. For my own part, I entirely agree with Trousseau, who was the first to give a decided opinion in this matter, when he says that the greater number of the cases of so-called congestive apoplexy are to be regarded in reality as rare, one might say anomalous, forms of epilepsy. At any rate, I can say, for the majority of the cases of this kind that have come under my own care, that after being subjected to careful and prolonged observation, they have shown themselves to be cases of unquestionable epilepsy.

For the arguments in support of this view, we refer the reader to the chapter on Epilepsy.

Among the disturbances of the sensorium is to be classed another symptom, one of the commonest in hyperæmia, namely, *dizziness.* It is rarely wanting, is, in many of the lighter cases, one of the most prominent symptoms, one also which is among the most distressing to the patient, and occurs as one of the prodromata—where such exist—of the severer forms of the disease. We would at the same time mention that this symptom seems to us to belong rather to the cases of active congestion than to those of passive hyperæmia.

The *disturbances in the sphere of the special senses*, also exceedingly common, are, in kind, identical with those which attend anæmia. Here, as there, the optic and auditory nerves are the ones affected, as is indicated by the appearance of specks and sparks before the eyes, temporary darkening and dimming of the visual field, hallucinations of sound, ringing and buzzing in the ears, diminution of the acuteness of hearing. There is sometimes, in cases of congestive hyperæmia, an abnormal sensitiveness to visual and auditory impressions, such as a bright light and the like, but whether it is due to excessive irritability of the peripheral apparatus or of the sensorium is uncertain. It is to be remarked that although one or the other of these affections of the nerves of special sense is almost invariably to be met with in hyperæmia, no matter of what variety or intensity, a complete loss of function—analogous to the amaurosis of anæmia—never occurs, or at least no such cases have come to our notice.

Disturbances in the sensitive sphere are—except for headache—of rather rare occurrence in hyperæmia. Now and then such patients complain of a temporary sensation of "going to sleep" or of formication in one extremity, or in the region of distribution of a certain nerve. Hammond claims to have demonstrated, with the æsthesiometer, paræsthesia, increase in size of the circles of dispersion. Real pain is complained of only in the head. Yet even headache is not always present. The patients often complain of a dull, scarcely to be called painful, sensation of confusion in the head.

The pain, as a rule, is of moderate severity, and distributed uniformly over the skull; it happens but rarely that it is unbearably severe.

Disturbances in the motor sphere, irritative as well as paralytic in character, are also met with in cerebral hyperæmia.

Unfortunately, owing to lack of sufficient help from experimental physiology, the exact relation of these symptoms to distinct pathological states is not so clearly understood as it is when they accompany anæmia. Omitting familiar preliminary descriptions, we will speak first of convulsions. *Most common* are the cases in which, among the regular symptoms of active congestion (marked vascular injection, tinnitus aurium, dizziness, sense of slight confusion, etc.), localized, momentary twitchings of the muscles of the face, or of one of the extremities, occur.

Next in order are those cases where, after the lesser symptoms of congestion have come and gone, sometimes indeed all of a sudden without their occurrence, either with or without an external cause, the patients become suddenly comatose (apoplectic), and are attacked with universal, typical, epileptiform convulsions, after which they either remain for a time somnolent or in a state of great mental confusion, or they fall into an almost maniacal condition, from which they gradually recover. *Finally*, a class of cases has also been regarded as cerebral hyperæmia, where the patients have been seized with general convulsions *unattended* with loss of consciousness (Andral and others). That convulsions of the first and second variety may arise from cerebral hyperæmia, we consider to be beyond question. We are, however, just as fully convinced that many of the cases, which are considered, in practice, and cited in literature, as being of this kind, are really cases of epilepsy ; and for the convulsions of the third variety, we would regard the diagnosis of simple cerebral hyperæmia as very exceptionally admissible (compare the following chapter, on Pathology). For the differential diagnosis between epilepsy and simple cerebral hyperæmia, we would refer the reader again to the chapter upon Epilepsy.

Paralytic symptoms are rare, though they do occur now and then. In the lighter cases they are entirely wanting, unless the temporary sensation of general feebleness is to be regarded as of this nature, a condition that, in fact, seems to point rather to enfeeblement of the power of innervation than to a disturbance within the motor nerve tracts.

In case of more violent attacks, a patient sometimes complains of a certain weakness and a feeling of heaviness in some of the fingers in one extremity or in an entire half of the body; most frequently of all it happens that there is a certain awkwardness in the use of words, or even, for a moment, a complete loss of the power of speech. Along with these motor disturbances are found the sensitive disturbances already mentioned, a feeling of formication and numbness. These symptoms, as a rule, pass away in the course of a few hours or days; but they may, on the other hand, develop into a complete hemiplegia. Rochoux, Rostan, Andral, Graves, and many others have recorded such cases, and they are in fact not so very uncommon.

It is scarcely possible to suppose that hemorrhage could have taken place in cases where the paralysis disappears even after a few hours, and especially when it passes away rapidly, as Graves describes; furthermore, the results of autopsies directly contradict such an assumption. Now and then, indeed, extraordinary cases are reported, where, in apparent contradiction to physiological laws, hemiplegia occurs without any loss of consciousness, and others where the autopsy shows a perfectly equal congestion of both hemispheres, an anatomical condition the reverse of what we should expect to find. We instinctively ask in cases such as these, which at best are rare, whether something was not overlooked in the autopsy. We append, in illustration, a case of this kind observed by ourselves, where the diagnosis is to be made only with the greatest difficulty:

M. K., four years old; a very delicate child. One aunt suffers from migraine; otherwise no marked hereditary tendency to any nervous disease.

Health good in early life. A short time ago it had an attack of erysipelas on the left thigh, with enlargement of the glands, in consequence of a slight erosion, ending four weeks ago, however, in complete recovery. On November 17, 1873, the patient walked out of doors, complaining only of suffering to an unusual degree from the cold. After returning home, it seemed as well as ever, until after three hours, when it began to behave strangely, became silent, speaking only a few words, and, finally, towards half-past six o'clock fell into a state of complete coma.

Immediately afterwards its father (himself a physician) discovered that the right arm and leg were completely paralyzed. At the same time convulsive movements came on, which will be described more exactly hereafter, lasted twenty minutes, disappeared, returned about a quarter of an hour later, again disappeared, to return a third time, the whole occupying about an hour and a half.

When I saw the child, at about eight o'clock, it lay on the back, in complete coma, or, at any rate, not responding to calling, pinching, etc., with head and face turned somewhat to the left, and both eyes turned likewise to the left. The left pupil was greatly dilated, the right apparently of normal size, or below. From time to time the eyes were rotated as far as to the median line, but never to the right, then to return immediately to their previous position.

There was no facial paralysis. The extremities on the right side lay quite relaxed (when not in convulsive action), and, when raised, fell like dead-weights again; those of the left side offered the normal resistance.

With momentary intervals of rest, the muscles of the right side of the face and the extremities of the right side were the seat of constant convulsive twitching, half choreic, half clonic, in which also the muscles of the right side of the trunk took part. The length of the intermissions was about half a minute. After the third attack, which lasted about twenty minutes, the convulsions ceased completely; the patient opened its eyes, as if startled, and appeared to hear calling. Quarter of an hour later it moved the right leg, though with difficulty.

During the convulsions the action of the heart was very rapid; afterwards normal.

No disease of the heart or lungs was to be found.

On the following day (18th November) the child was mentally perfectly clear, and quite merry. The movements of the right leg and the right arm were perfectly normal; but the pressure with the right hand was feebler than with the left. On the following day no symptoms remained which could recall the severe attack through which the patient had passed. Up to the present time—a year later—there has been no recurrence of the symptoms, and the child has, in fact, been perfectly well.

Besides the conditions described, others are occasionally met with which differ greatly from each other, and may be present or not, according to the particular form and cause of the hyperæmia. The most prominent among them concern the *circulatory apparatus*, and, indeed, properly speaking, a case of cerebral hyperæmia is incomplete without them. The greater number of these disturbances of the circulation stand, etiologically, on the same footing with the cerebral symptoms; exceptionally only are the former secondary to the latter.

In cases of active congestion the patients complain, though not invariably, of palpitation of the heart, accompanied with a sense of oppression; the impulse of the heart is often increased in strength, and its action more rapid; the pulse is, therefore, as a rule, more rapid, fuller, and more resistant; the carotids pulsate strongly. The face becomes reddened, and a sense of heat rises to the head. It deserves, however, to be expressly mentioned that this vascular excitement and congested face are not always present, even in cases of active hyperæmia. In cases of passive hyperæmia, of course, a totally different state of things prevails, the veins being overfilled, and the face swelled and cyanotic. The character of the pulse and the heart's action will manifestly differ in accordance with the etiological moments of the case.

We would refer in this place to a variety of cerebral hyperæmia which has been described as the *febrile form.*

Among adults we have never seen a case which would have necessitated such a diagnosis.

Such cases suffer, according to the accepted description, from headache, delirium, fever (hot, dry skin, thirst, febrile urine), and from constipation. The symptoms gradually subside after some hours or days.

Whether this state is to be regarded as simple cerebral hyperæmia, as is done even by so careful an observer as Hasse, seems to us to be a question which is by no means definitely settled.

With children this febrile form of cerebral hyperæmia is believed to occur much more often. In addition to the symptoms described, it is said that there is often somnolence, and that the attack begins with vomiting, sometimes even with retardation of the pulse. Bouchut describes cases of this kind, resembling meningitis, as "congestion-neuroses of the brain." Are they really to be properly so classed? In the first place, it seems to us to be by no means certain that there was really elevation of the general temperature of the body in all these cases, for the figures are given but rarely; and Reynolds, indeed, describes the extremities as cold, and only the head as hot. Further, it is far from being proved that hyperæmia, confined to one portion of the body, can excite a condition of general fever, although we admit that possibly the case may be different with hyperæmia of the central nervous system, involving some disturbance of the heat-regulating centres.

While we, therefore, would not deny that the existence of a febrile form of the affection is possible, yet we hold that further proofs are required for its establishment as a matter of fact.

The *respiration* in cerebral hyperæmia differs according to the form and origin of the disease, without offering anything characteristic. We would emphasize only the snoring, stertorous breathing which is peculiar to the comatose forms, and is just like that which occurs in hemorrhagic coma.

Certain other symptoms are sometimes met with that are not to be distinguished from those which occur in various other cerebral diseases (meningitis, hemorrhage), namely, vomiting and *constipation.* Neither is apt to be severe; but severe nausea and vomiting, when they do occur, indicate that there is a relatively dangerous intensity of the disease.

This would be the proper place to speak of the circumscribed local hyperæmias. These conditions, so far as they occur in connection with the severer intracranial diseases, tumors, softening, thrombosis of the sinuses, etc., will be treated of elsewhere. They are met with in their pure form only in consequence of *ligature of the carotid*, where the blood flows to an abnormal degree into the opposite half of the brain.

It is striking how seldom, under such circumstances, abnormal symptoms occur, or, to say the least, have been reported. Only here and there it is stated that the patients, immediately after the operation, were seized with trembling and convulsive twitching in the extremities of the same side with the ligature, lasting for several hours.

The paralysis on the same side, reported occasionally, ought scarcely be referred directly to the hyperæmia (autopsies are wanting), since it has come on, at the earliest, after some days, sometimes only after several weeks.

Pathology.

It is self-evident that the influences which underlie the morbid symptoms due to increased vascularization of the brain must be of widely different kinds, according as the case is one of active, arterial, or of venous hyperæmia. In the former condition the following are probably the important moments:

first of all, an increase in the intracranial pressure, directly demonstrable in the case of young children by increase in the tension of the anterior fontanelle; next, the assumption of Andral's certainly deserves consideration and further testing, namely, that in certain forms of active hyperæmia, especially the plethoric forms, an increased supply of red blood-globules is able of itself to excite cerebral symptoms; finally, it appears to us not unlikely that, in consequence of being furnished more abundantly with the nourishment brought by the arterial blood, the functional activity of the brain may become more energetic.

In the case of venous hyperæmia the moments chiefly of consequence are the following: first, the increase—at times very great—of the intracranial pressure, which, as mentioned above, can be directly detected in infants by observing the fontanelle, and which Jolly succeeded in producing experimentally by compression of the jugular veins; secondly, a venous stasis of any amount is likely, in the manner referred to at the outset, where the conditions regulating the cranial circulation were discussed, to bring about a diminution in the arterial supply, so that, in fact, physiologically considered, venous hyperæmia is equivalent to arterial anæmia. This fact suggests at least an explanation of the similarity in some respects between the symptoms, whether clinical or produced experimentally, of cerebral anæmia and hyperæmia. It is self-evident, however, that this statement concerns not the cases of arterial congestion, but only those of venous stasis. Finally, one moment is to be emphasized, which has lately been left out of account, namely, the insufficient removal of carbonic acid, which must necessarily attend the obstruction to the flow of venous blood, from which follow the results due to the action of carbonic acid on the different parts of the brain.

How, and in what degree, these different influences will act in any special case, it is often impossible to say, and the want of space prevents us from dwelling upon them all in this place.

For the action of arterial anæmia we would refer to the chapter on that subject, and for the effect of the increase in intracranial pressure to the chapter upon Cerebral Hemorrhage. Certain points alone shall detain us for a moment.

The cause of the symptoms of mental excitement, which are to be observed almost exclusively in the cases of active congestion, is to be sought in the more rapid supply of nourishment to the brain ; that of the symptoms of depression, even amounting to coma, in the increased pressure.

The general convulsions are probably due to the anæmia, produced indirectly in the manner explained above. Since they correspond, according to experimental testimony, to the most extreme degrees of hyperæmia, and, on the same evidence are shown to be associated with symptoms of disturbance of the cerebrum proper (which is much more readily affected than the convulsion-centres by changes in the circulation), namely, in the case in hand, with coma, it seems to us quite plain that the so-called variety of hyperæmia, which is characterized by convulsions without coma, is no hyperæmia at all.

Course—Prognosis.

It has been made evident, by the remarks on symptomatology, that very great differences exist, in point of severity and duration, and consequently in the prognosis as well, between the different forms of the affection. Special cases differ so greatly among themselves, that general rules for prognosis cannot be drawn from them. The venous hyperæmias, brought about by temporary causes (straining at stool, etc.), are almost without significance ; now and then, but very rarely, obstinate attacks of coughing, especially in tussis convulsiva, give rise to more threatening symptoms. The slighter forms of congestive hyperæmia also are rarely dangerous in themselves. At the worst, the attack leaves behind it a temporary headache, and a certain degree of mental discomfort precedes for a while the return to complete health. At the same time, the frequent return of these attacks is not to be made light of, although it is true that they may recur for years without permanently bad results ;[1] thus, in the case described above, the patient was in perfect health during the intervals. On the other hand, it happens not unfrequently that repeated attacks of cerebral congestion are followed eventually by hemorrhage; and it is the dread of this which has given the affection a bad name among the laity. The anatomical results which follow frequent attacks of arterial, as well as long-standing venous congestion, have been described above. It is very difficult to decide just what are the clinical symptoms which are due to these vascular dilatations or to the atrophy of the brain; we would refer the reader to the chapters where these subjects are discussed. At any rate, the fact that these anatomical changes do sometimes follow, indicates that the occurrence of these lighter hyperæmias is to be regarded as a serious matter.

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[1] The typically rhythmical cases, an instance of which is described by Andral, are doubtless to be regarded, as Hasse says, as forms of undeveloped intermittent fever.

The prognosis in the severer forms is much more grave: such are those attended with delirium, and the apoplectiform congestion, and the other varieties described above. They may indeed threaten life with immediate danger. Cases have unquestionably occurred—they are reported in the writings of authors from the time of Boerhaave and Morgagni to our day—where death has taken place, preceded by the symptoms of apoplexy, and where, at the autopsy, not a trace of extravasation, only a high degree of cerebral hyperæmia alone, was found.

The frequency with which this occurs has, to be sure, often been exaggerated. Where death is, however, really brought about by simple hyperæmia, it takes place during coma. In those cases where death has been observed to follow delirium, it has been preceded by loss of consciousness.

The prognosis with regard to complete recovery is much more unfavorable than with regard to the preservation of life. It is certain that some forms of the affection may end in complete restoration to health (we shall refer to them again directly, in speaking of the treatment); but in other cases the original cause of trouble is not to be removed.

Treatment.

The treatment, of course, aims both at overcoming the causes of the disorder and at relieving the individual attacks. With regard to the former object, we must refer to the descriptions of the causes of cerebral hyperæmia, given in another part of this work. It is plain that this prophylactic portion of the treatment must vary according as we have to deal with general plethora, organic heart disease, vaso-motor disturbances in hysterical persons, or collateral congestions around a focus of disease. Through the treatment of these fundamental conditions, as far as it is possible, the attacks of hyperæmia may sometimes be warded off.

Even with regard to the attacks themselves, however, the cases have to be considered on their individual merits. For general rules we would mention only the following: the patient must lie with the upper part of the body raised, and it is advisable also that the arms should be stretched upward (as is done in epistaxis, and for similar reasons). Further, he must remain quite at rest, making no movements. Useful as exertion may be at times as a prophylactic measure, yet during the attack itself, whether it be one of arterial congestion or of venous stasis, it can only do harm, since it causes more violent action of the heart, which in the former case directly aggravates the congestion, in the latter increases still more the intracranial pressure, already above the normal. These rules generally cover the necessities of the light, passing forms of the disorder, while the severer forms frequently demand more energetic measures.

The *treatment by drugs* seeks to diminish the quantity of blood circulating in the cranium, either by the direct withdrawal of fluid, or by reducing the amount of blood-serum in the cranial cavity through artificially increasing the circulation in other parts of the body, or through some direct action on the vessels of the head. Each of these methods has its advantages; under certain conditions both may be combined together.

In spite of the theories of Kellie, excellent practitioners, such as Abercrombie, for instance, even though, in general, they supported these very views, have had no doubt as to the efficiency of *blood-letting* in cerebral hyperæmia; the evidence afforded by the diminution in the severity of the symptoms, which follows, for example, an accidental nose-bleed under these conditions seeming to point too clearly to the propriety of this treatment; under certain conditions we could not get on without using it. To be sure, the use of general venesection has become constantly more and more limited; so that, whereas formerly it was the invariable treatment in every case of cerebral hyperæmia, especially of the congestive variety, it is to-day, and with reason, employed but rarely. In most cases of venous stasis it is not only unnecessary but is even injurious, just as any influence which diminishes the functional activity of the heart, under the conditions which obtain in this state, must be injurious. Only when life is immediately endangered by the cerebral pressure or the overloading of the blood with carbonic acid gas, can it be necessary. In the lighter forms of the active hyperæmias, it is not, as a rule, injurious (unless in the case of hysteric or chlorotic persons), but it is usually unnecessary. Only when the severer symptoms are present (delirium, *coup de sang*) is it needful to resort to the lancet. General indications for its use are furnished by the presence of marked *turgor faciei*, strong cardiac impulse, and abnormal fullness and tension of the arteries. The amount of blood to be drawn, as well as the question of the future repetition of the operation must be determined by the symptoms in the particular case. Rules of universal application cannot be given. Under certain conditions, where moderate symptoms are present, which nevertheless do not yield to other treatment, local blood-letting must be used, leeches being applied over the mastoid process, or cups at the back of the neck. This is the only variety of the treatment which is applicable to children, and is generally the one to be used with aged persons.

In the lighter cases, where blood-letting is not called for, the so-called derivative treatment may be employed—which, indeed, in the severer forms is always to be used as an adjuvant to the other. Its aim is to attract the blood away from the head and rather towards other parts of the body, or else to diminish the quantity of its fluid constituents.

The former indication is met by the use of irritating foot-baths (made with lye, mustard, etc.), or of any means capable of exciting cutaneous hyperæmia. As a rule, the quickly working irritants, such as mustard and vinegar, or rubbing, are the most applicable. They may be used on the skin of the upper and lower extremities. *Purgatives* play an important part in the treatment, exerting, as the phrase is, "a derivative action in the intestines;" but their chief use is probably in promoting watery discharges.

Out of the great number of cathartics at our service, those are generally chosen which produce the effect mentioned in the quickest possible time. These are the salines, croton-oil, colocynth; irritating *enemata* have a similar action.

The production of diuresis (by saline diuretics) is also of importance in obstinate cases. Reynolds[1] says that an abundant discharge of urine will sometimes have a favorable effect on the cerebral symptoms in cases where blood-letting and purgatives have been tried in vain.

In cases of congestion with great sense of heat and headache, marked *turgor faciei*, the application of cold to the head plays a useful part in the treatment; in the lighter cases, cold-compresses suffice; in the severer, ice-baths are necessary. Whether these applications really cause a contraction of the blood-vessels in the brain or not, is doubtful; but certainly they sometimes diminish the subjective sense of heat and the headache. Cold douches on the head very likely increase temporarily the power of reaction of the system in congestive apoplexy, but cannot be expected to remove the hyperæmia altogether; on the contrary, they are found in practice to be too irritating, and their final effect is an increase of the arterial congestion. They are therefore to be avoided under such conditions, and their use is to be restricted to cases of coma from venous stasis.

On the other hand, in many cases of chronic arterial congestion the *cold-water treatment* is often beneficial. For its success it is absolutely necessary that it should be applied systematically at an institution for the purpose. Experience has taught that, unless this can be carried out, more harm will often be done than good.

Finally, it need scarcely be mentioned that, in active hyperæmia, the *diet* should be carefully attended to. Above all, all substances which increase the action of the heart are to be avoided, especially the alcoholic drinks, tea, and coffee. In case of venous congestion from disease of the heart or lungs, on the contrary, these agents are occasionally indicated, as will be pointed out in speaking of these affections.

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[1] Article "Congestion of the Brain," in his System of Medicine.

It will of course be a matter for the skill and tact of the physician to determine whether in a particular case the cerebral congestion is due to suppression of normal or pathological secretions; such as hemorrhoidal discharges, menstruation, and the like. We wish only to direct attention to this point here in passing; since to discuss it here at length would carry us too far out of our way.

CEREBRAL HEMORRHAGE.

A full list of the earlier works on this subject will be found in *Joseph Frank's* treatise, entitled: Die Nervenkrankheiten. Leipzig, 1843. Uebers. von Voigt, im Capitel: Der Schlagfluss, 1. Bd. pag. 283, ff.—The works on diseases of the nervous system enumerated at the beginning of this volume may also be consulted. In addition to these treatises and to those quoted in the text, the following works may be enumerated: *A. Gubler*, De l'hémiplégie alterne, etc. Gaz. Hebd. Paris, 1856.—*Bouchard*, Des dégénerations secondaires de la moëlle épinière. Arch. Génér. 1866.—*A. Eulenburg*, Ueber den Einfluss von Herzhypertrophie und Erkrankungen der Hirnarterien auf das Zustandekommen von Hemorrhagia Cerebri. Virchow's Arch. Bd. 24.—*Ch. Bouchard*, A study of some points in the pathology of cerebral hemorrhage; transl. by Maclagan. London, 1872.—*J. L. Prevost*, De la déviation conjuguée des yeux, etc. Paris, 1868.—*Charcot*, Leçons cliniques sur les maladies des vieillards. Paris, 1867.—*Hughlings Jackson*, in R. Reynolds' System of Medicine, II. ed. Art. Cerebral hemorrhage and apoplexy, pag. 521, et seq.—*Bourneville*, Etudes cliniques et thermometriques sur les maladies du système nerveux. Paris, 1872. I. fasc.—For *Meningeal apoplexy*, consult the works enumerated under the head of Pachymeningitis interna hemorrhagica. Of the remaining works, the most important are quoted in the text. Finally we must add: *R. Lépine*, Note sur deux cas d'hémorrhagie sous-méningée. Paris, 1867. 39 SS.

A.—Intra-Cerebral Hemorrhage in General.

The different forms of intra-cranial hemorrhage vary widely, both in respect to their causation and to their pathological anatomy. They may arise from external injuries to the head, even though the bones of the skull itself may not be affected, or they may originate in affections of the cerebral membranes, especially in cases of disease of the dura (pachymeningitis hemorrhagica). The bursting of aneurisms attached to the larger arteries, such as are particularly common at the base of the brain, may give rise to profuse bleeding. The same thing may occur as a result of the venous stasis which follows thrombosis of the sinuses. These forms will be treated of in another part of this work.

But even among the intra-cerebral hemorrhages there are certain ones which would be discussed more properly elsewhere than here; such are the hemorrhages occurring in the neighborhood of certain tumors, or in parts where softening or inflammatory changes have already taken place.

We shall have to do for the present only with the intra-cerebral hemorrhages proper, the spontaneous or primary forms, as they are also called, which follow from other causes than those described.

Historical Sketch.

It was until recently customary, and is still to some extent, to speak of cerebral hemorrhage by the name of one of its symptoms (to be sure, one of the commonest and most striking), namely, "*apoplexy*." This practice deserves to be abandoned, with the same right that that of Hippocrates, followed even by J. P. Frank, and, on the whole, the more intelligent of the two, has been abandoned.

Up to the time of Frank, apoplexy was divided into local and general apoplexy; the former being also known as paralysis. The term apoplexy denotes, however, in fact, only a clinical symptom ("proprie notat percussionem"), and it is to be understood to-day in the sense in which Boerhaave originally defined it, and in which Cullen, Stoll, and others in the last century further explained it: "Subitanea integra actionum, sensuum tum externorum tum internorum omniumque motuum voluntariorum abolitio, superstite pulsu sæpe forti et respiratione magna difficili stertente, una cum imagine somni perpetui atque profundi."

With this definition in mind, our fathers held that apoplexy might arise from many causes, just as we to-day speak of it as occurring not alone in consequence of extravasation of blood, but as a consequence of embolism and various other morbid processes.

That cerebral hemorrhage (in the present sense of the term) was to be classed among the causes of apoplexy, was first pointed out distinctly by Wepfer; the belief was elevated to the rank of a scientific fact through the efforts of Valsalva, Morgagni, Fr. Hoffmann, and especially of Boerhaave and his school, their arguments attracting finally so much attention that gradually the present tendency to confound together and interchange the names of these two processes arose.

The older physicians, having only a clinical acquaintance with apoplexy, distinguished between several varieties of the affection, and endeavored to trace out the symptoms by which they could be identified in practice.

In this way they came to speak of apoplexia sanguinea, serosa, nervosa, and ex inanitione. We have already spoken of these conditions in discussing cerebral anæmia and hyperæmia. We can now see plainly that the diagnostic endeavors of our forefathers, who sought to learn the causes of the apoplectic state from examination of its symptoms alone, were doomed to fall short of their mark in many cases, and that they are in fact unnecessary, since we no longer believe that distinct diseases exist corresponding to these names, preferring to mass together the various clinical phenomena under the term *apoplectic attack*, in the sense given above.

It is difficult to follow the different phases in the development of the doctrine of cerebral hemorrhage, as it arose gradually under the combined labors of many observers. Even up to 1820, according to Joseph Frank, more than two hundred and fifty writers had described the "apoplectic stroke," and since then the literature of the subject has increased to an incredible extent. For this reason we shall select out of this mass of material only the following fragments, to which the views entertained at the present day lend a special interest.

With reference to the etiology of the disease, Testa, Corvisart, and Kreyssig were the first to notice the connection between cerebral hemorrhage and disease of the heart; since which time this connection has never been lost sight of. Abercrombie and Rokitansky, but especially Virchow and his school, laid stress upon the significance of diseases of the cerebral vessels. The most important investigations in this direction in later times are those of Charcot and Bouchard.

In the matter of working out the anatomical details of the affection, the older French school was particularly active in the beginning of this century; later, investigations in this direction began to be made on every hand.

The credit of having first studied the subject carefully and thoroughly from the point of view of symptomatology, belongs, among the French authors, to Rochoux, and next, to Andral; among the Englishmen, to Abercrombie and Todd. The study of the localization of pathological changes falls within the last decade.

The history of the treatment of cerebral hemorrhage is shorter than that of any of the other departments of the subject; we can claim, in fact, to have made but little, if any, advance in this field. Even the important change which the introduction of electro-therapeutics has made in the treatment of nervous diseases in general has been but slightly felt in that of this particular class of cases.

Etiology.

We do not deem it worth while to discuss all the theories which have been maintained from time to time as to the causes of cerebral hemorrhage, being taken up only to be again abandoned. Only two of them demand a brief notice, since they have been held by writers of repute, even within our own time. These two theories have this in common, that they regard a change in the cerebral tissue as the primary factor, and the hemorrhage itself as secondary to that.

Rochoux was of the opinion that, in the first place, a process of softening usually occurs, and then the extravasation (ramollissement hémorrhagipare); but neither he nor Todd, who adopted the same view, undertook to define the nature of this softening more exactly. At present it is known that this prehemorrhagic softening—the existence of which indeed was disputed by Durand-Fardel and by Gendrin—is of secondary nature, the result, partly of the imbibition of blood serum, partly of inflammatory reaction of the tissues around the extravasation, and, finally, in all probability partly of a partial retrograde metamorphosis of the damaged nerve-fibres (Bouchard). If, however, it is not true that hemorrhage occurs exclusively as a result of softening, still there is no doubt that in cases of thrombosis and embolism such a connection may exist. These forms of the affection do not belong in the same category with the proper, genuine varieties of cerebral hemorrhage, and we would refer the reader for a further description of them to the special chapters upon those subjects.

The doctrine, espoused by Calmeil, that the cerebral hemorrhage is nothing else than a hemorrhagic inflammation (he calls it encéphalite locale aigue avec caillots sanguins), can hardly—taken in his sense—find many defenders to-day.

Still less foundation is there for the view that a primary *atrophy of the cerebral substance* is to be reckoned among the causes of hemorrhage. The essential argument against this theory is well given by Hasse, in his statement that the cerebral atrophy goes on much too slowly to give rise to marked congestion of the brain. The reduction in the size of the brain is made up for in many cases, to a certain extent, by a thickening of the bony skull (Paget), or by an increase in the size of the frontal sinuses. The part played by the cerebro-spinal fluid in this process of compensation is, moreover, much more important than that taken by the circulating blood, as is proved by the relatively greater dilatation of the perivascular spaces, under these circumstances, than of the blood-vessels. We agree, further, entirely with Hasse in his opinion, that the frequent concurrence of hemorrhage and atrophy is probably to be explained by the fact that both are affections of advanced age.

Two conditions are universally recognized at the present day as being among the most important of the predisposing causes of genuine cerebral hemorrhage.

1. Increase of blood tension, especially in the vessels of the arterial system.

2. Disease of the walls of the vessels themselves. We would preface the description of the details of these conditions by a few general observations.

If we except the cases where bleeding is due to injuries or ulcerations (gastric and typhoidal ulcerations, and the like), and those caused by diseases of the vessels, such as are familiar and easily to be demonstrated, it may be said that hemorrhages occur almost exclusively in one of three vascular regions—namely, the lungs, the mucous membrane of the nose, and the brain. It is well known to be still a matter of doubt whether hæmoptysis can occur except in connection with diseases of the lungs. It rarely attends a temporary increase in the arterial pressure, except in persons with a predisposition to phthisis, scarcely ever in healthy persons. Finally, it need scarcely be pointed out at length how very rarely it happens that venous stasis in the lungs, even if severe, gives rise to pneumonia, and even then it occurs only with the aid of enormous temporary increase in the pressure. Increase in blood pressure of itself leads then seldom or never, in a previously healthy person, to pulmonary hemorrhage.

Even epistaxis, in consequence of increase in the blood pressure alone, is a thing of the rarest occurrence. It is true that it is sometimes met with in connection with chronic disease of the kidneys. It may well be asked, however, whether in such cases alterations of the walls of the blood-vessels are not present at the same time, a possibility to which the recent investigations of Gull and Sutton have lent increased support.

To be sure, bleeding at the nose is often observed in many, especially youthful persons, in consequence of the slightest increase in the action of the heart; but for this very reason it seems not improbable that in these cases changes in the vascular walls are present (though their nature is entirely unknown). In the case of healthy persons in adult life, simple increase in the arterial blood pressure, however great, is incapable of producing such an effect.

If we bear this fact in mind, and remember, further, that rupture of blood-vessels almost never occurs in any other organ of the body as a consequence of elevation of the blood pressure[1]—indeed that this result is of the rarest occurrence even in the brain in cases of contracted kidney (vide below)—it seems to us entirely fair to ask, *how is it possible that increase in arterial pressure should cause hemorrhage in the brain alone? Is it really true, as is generally believed, that the local diseases of the cerebral vessels, and the increase of tension in the vascular system, are influences which tend with equal force to bring about cerebral hemorrhage; is not the former influence rather to be regarded as of principal, and the latter only as of secondary importance?* Judging from the facts which lie before us, we are compelled to decide that the latter answer is, for the vast majority of cases, the correct one.

The most important predisposing cause of cerebral hemorrhage, then, consists in diseases of the cerebral vessels, especially the arteries. In the last twenty-five years this fact has been quite universally recognized, and, further, this diseased condition has been supposed to consist essentially in a sclerotic change of the arterial walls.

The question was then raised, however, how it was that in other parts of the body sclerosis of the walls of the vessels gives rise to vascular rupture only after aneurisms have formed, whereas in the brain the extravasation seemed to come from vessels without aneurisms. Charcot and Bouchard have recently explained this apparent paradox by the demonstration of the so-called miliary aneurisms. These formations had, to be sure, already been observed in single instances, before this time, by Cruveilhier[2] and Virchow,[3] by whom they were called "saccular dilatations" (ampulläre Ektasien), as well as by Heschl[4] in the pons Varolii, and by Meynert;[5] but Charcot and Bouchard were the first to show that the "miliary aneurisms" occur *regularly* in the cases of spontaneous hemorrhage, so-called (out of seventy-seven cases they discovered them in every one), and that the hemorrhage actually takes place in consequence of the rupture of these formations.

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[1] I learn by letter from my respected friend, Prof Leber, in Göttingen, that it is very questionable whether the *retinal hemorrhages*, which occur, as is well known, *in connection with contracted kidney*, are to be referred simply to the high arterial pressure. On the contrary, it is probable that here also vascular diseases are concerned in the result. We cannot, of course, enter at length into the details of this matter.

[2] Anat. Path. du Corps Humain. Livr. XXXIII.

[3] Virchow's Arch. III. Bd.

[4] Wiener med. Wochenschr. 1865, 6 u. 9 Septbr.

[5] Allg. Wiener Wochenschr. 1864. No. 28.

Since then their statements have been confirmed by Vulpian, Barth, Béhier, Lionville,[1] and, on the ground of observations extending over several years, by Zenker[2] and also by Roth.[3]

These aneurisms are of reddish color, though varying in size from that of scarcely visible bodies to that of a pin's head (0.2–1 mm.), and are situated on the arterioles. Their number is very variable. Sometimes but few are found, in the vicinity of the ruptured vessel, sometimes they are scattered through the entire brain to the number of one hundred and upwards. The relative frequency of their occurrence in the different regions of the brain is quite constant; their favorite seat is the thal. opt. and corp. str., then the convolutions, pons Varolii, centrum ovale, middle cerebellar peduncles, pedunc. cerebri, and med. obl. are progressively less apt to be affected. This table of itself bears presumptive evidence to the importance of the miliary aneurisms as a cause of cerebral hemorrhage, since it corresponds with that showing the relative frequency with which the different regions of the brain are the seat of extravasation. It is further noticeable that in other vascular regions than the brain, these miliary aneurisms are only very exceptionally found, a circumstance which throws much light on the fact above stated—that the so-called spontaneous hemorrhages occur almost exclusively in the brain.

The miliary aneurisms are met with, not only in the case of persons who have already suffered from cerebral hemorrhage, but also in that of those who have died from other diseases. Such persons might be said to have had a tendency to hemorrhage. Quite rare before the fortieth year, these aneurisms are met with more and more frequently with advancing years; and in fact the liability to them increases at a more rapid rate than the age of the patient (the relatively smaller number of persons living at advanced ages being taken into consideration)—another respect in which their history corresponds with that of cerebral hemorrhage.

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[1] All referred to in the later writings of Bouchard.

[2] Tageblatt der Leipziger Naturforscherversammlung.

[3] Correspondenzblatt f. schweizer. Aerzte. No. 6.

The anatomical process which results in the formation of these aneurisms is believed by Bouchard and Charcot to be of the nature of chronic periarteritis. According to them, there occurs first a multiplication of nuclei in the lymph-sheaths and adventitia, sometimes with, sometimes without a thickening of the latter, often accompanied by simple atrophy of the muscular coat. In case the atrophy takes place without the thickening of the adventitia, the miliary aneurisms become formed, and from them—either in consequence of an increase of the blood pressure, or, where the thinning of the walls of the vessel has reached a certain point, even spontaneously, without any increase in blood pressure—the escape of blood takes place.

According to Charcot and Bouchard, the formation of these miliary aneurisms is not necessarily associated with sclerotic changes in the arterial walls. Besides claiming that the two processes which give rise to these results differ pathologically in certain respects, they maintain that in a quarter of the cases of persons who had died from hemorrhage, and in all of whom the aneurisms were found, it was impossible to detect any sclerotic change in the larger arteries.

Zenker admits the latter fact, but draws from his own investigations the conclusion that the inner coat of these miliary aneurisms, and of the arterial twigs bearing them, is found to be the seat of circumscribed thickenings, associated with the other changes, which are peculiar to the so-called arterio-sclerosis. According to the view of Zenker, therefore, the intra-cerebral aneurisms of Charcot and Bouchard resemble the larger extra-cerebral aneurisms which occur at the base of the skull in the very respects which in the case of the latter make them prone to rupture.

Long before the discovery of the minute aneurisms, which have just been described, the cause of cerebral hemorrhage was sought even by Abercrombie in arterial diseases of various kinds, among which the *atheromatous degeneration* held a prominent place. Without doubt this process, which need not be described here in detail, may give rise indirectly to hemorrhage, by leading to the formation of *aneurisms.* Such aneurisms are met with, generally, on the arteries of the base of the brain and the larger of their branches; but, at the best, they are not often seen. For this reason the cases of cerebral hemorrhage of this origin are not of common occurrence, especially as compared with those of so-called spontaneous origin, when the extravasation occurs in the midst of the brain from the miliary aneurisms. It is further to be mentioned that rupture of the larger aneurisms of the basilar, middle cerebral, and similar arteries, when it does take place, gives rise, as a rule, to hemorrhage into the meninges. (Vide the chapter on this subject.)

What answer shall we give to the question, whether simple atheromatous degeneration, with the brittleness and rigidity of the vascular walls which are its natural consequences, but uncomplicated by the formation of aneurisms, bears any definite causal relation to cerebral hemorrhage?

It was formerly believed that when this condition was found to be present in the arteries of the base of the brain, it was fair to assume its existence also in those of the interior, and to refer the rupture to its influence.

In view, however, of the fact, that hemorrhage occurs, and not unfrequently, without its being possible to discover the least trace of morbid change in the larger vessels, and that, on the other hand, in other vascular regions, in spite of the presence of pronounced atheromatous changes, spontaneous hemorrhage is never (unless aneurisms are also present) to be met with, it may be fairly concluded that atheromatous degeneration does not of itself directly lead to rupture.

In making these statements, however, we would by no means be understood to deny its indirect influence in this direction, which it owes mainly to the fact that it causes an impairment of the normal elasticity of the walls of the larger vessels, so that they cease to check the force of the pulse-wave to the normal extent, in consequence of which the blood pressure in the arterioles (the seat of the miliary aneurisms) of necessity becomes abnormally great.

The admission of these facts brings us to a conclusion of great diagnostic importance. It has been the habit in cases of apoplexy to attach great weight to the condition of the peripheral arteries (radial and temporal), in judging whether the case in hand was one of cerebral hemorrhage; it follows, from what has been said, however—and indeed clinical experience teaches us often enough the same lesson—that the existence of rigidity of the peripheral arteries is of no diagnostic significance. Although it is true that in such a case we have a right to assume that sclerosis of the larger cerebral arteries is present, yet, as we have seen, this does not of itself lead to hemorrhage; and, on the contrary, we have learned that the conjunction of miliary aneurism with hemorrhage is quite common without there being the least trace of change in the coarser vessels. At the best, then, confirmatory evidence is all that the presence of this latter condition can furnish.

With regard to the various other diseased conditions of the cerebral vessels, we can deal with them very summarily. Paget was the first to speak of the occurrence in them of *fatty degeneration* and its significance as a cause of hemorrhage, though, even before him, Rokitansky, Koelliker, and Hasse had described analogous processes under other names; Wedl, Leubuscher, Virchow, and others, have also busied themselves with this question. Bouchard, as well as Hasse, points out—and, in our opinion, with justice—that the evidence in favor of this view is by no means so strong as it formerly appeared. For example, we would call attention to the fact that this condition of fatty degeneration is found at all ages, and, indeed, among cachectic children even more frequently than among aged persons, and, on the other hand, that, in many cases of death from hemorrhage, not a trace of it is to be found.

The dissecting *aneurisms*, described by Koelliker,[1] Pestalozzi,[2] Virchow (l. c.), are of secondary importance; and the anatomical details of their formation, in part still the subject of controversy, must be passed over here in silence. Though it cannot be denied that such aneurisms may now and then give rise eventually to hemorrhage, yet the cases are of exceptional occurrence. Supposing the view entertained by Zenker, as to the origin of the miliary aneurisms of Bouchard, to be correct, however, the question might fairly be raised, whether there may not be a regular transition from this to the dissecting form of dilatation.

We turn, now, to another important part of the history of the pathogenesis of cerebral hemorrhage, namely, to the *changes in*

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[1] Zeitschr. f. wissenschaftl. Zoologie. I. Bd.

[2] Ueber Aneurysm. spur. der kleinen Hirnarter. Würzburg, 1849.

the pressure of the blood. It is a matter of daily experience (and it is therefore unnecessary to adduce statistics to prove it) that hemorrhages may occur without the least elevation of pressure. Every physician has met with instances where persons in whom no abnormal condition of the circulatory apparatus existed, and who, without being exposed to any influence which could excite the action of the heart, during absolute rest, perhaps during sleep, have nevertheless been attacked with hemorrhage. This is, to be sure, not the usual history, but even its occasional occurrence proves that increase of blood pressure is not an essential and necessary link in the chain of causes, but exerts only an accessory influence.

In cases of this kind it is to be supposed that the degeneration of the aneurisms was so far advanced that even the normal blood pressure sufficed to bring about the rupture.

If it had been made certain that increase of blood pressure without local disease of the arteries could of itself give rise to vascular rupture, then, to be sure, these two influences would have to be regarded as of equal significance, though not present with equal frequency. This is, however, not the case, or at least it has not been proved by arguments which satisfy all the requirements of modern science, for the older observations, made at a time when the miliary aneurisms were not known and not sought after, hardly deserve to have much weight attached to them. Even at the present day, how rarely is a full allowance of time given to this search? Certainly, the investigations in this direction need to be re-made, especially in cases where hypertrophy of the heart exists in connection with contracted kidney, a condition which has been regarded as particularly favorable to the occurrence of cerebral hemorrhage, due to simple increase of blood pressure. It is to be remarked, to begin with, that the relative rarity of occurrence of the hemorrhage in these cases constitutes *à priori* evidence against the hypothesis in question. Frerichs[1] reckons cerebral apoplexy only among the accidental complications of Bright's disease. Out of two hundred and forty-one cases collected by him, it occurred but six times.

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[1] Die Bright'sche Nierenkrankheit. Braunschweig, 1851.

Among two hundred and fifty cases of Dickinson's, in which, to be sure, those of amyloid degeneration, etc., without increase of arterial pressure, are included, hemorrhage was found but seventeen times, and of these seventeen cases atheromatous disease of the arteries was present in fourteen; while Senhouse Kirkes,[1] out of thirteen cases of hypertrophy of the left ventricle, associated with cerebral hemorrhage, found disease of the arteries in twelve.

These considerations—which we have not space to discuss at length—certainly justify the opinion here maintained, that increase in arterial pressure, by itself, without disease of the cerebral arteries, gives rise but rarely to cerebral hemorrhage.

When, however, these local changes in the vascular walls exist, then the increase in pressure is unquestionably more active than any other influence in bringing about the rupture.

This increase in vascular tension may be either permanent or temporary. As productive of the former, hypertrophy of the left ventricle is to be particularly mentioned; but although it is true that this affection, and the increase of pressure dependent upon it, may lead to vascular rupture, it is not proper to speak without restriction of heart diseases in general as standing in a causal relation to cerebral hemorrhage, as the older writers used to do.

Eulenburg, indeed, points out with justice—basing his statements upon the facts first brought out by Traube—that only certain of the forms of hypertrophy of the left ventricle exert the influence in question, those, namely, which really are associated with increase in the arterial pressure—in other words, not those forms which follow on valvular diseases at the orifices of the heart, or from aneurisms in its neighborhood, but only those which attend contracted kidney and diffused arterio-sclerosis (we purposely avoid entering upon the disputed question as to which of these processes is primary and which secondary), and perhaps the spontaneous hypertrophy due to excessive exertion.

But, further, even where permanent elevation of pressure exists, it is often unable, for the very reason that it was only gradually brought about, to cause vascular rupture, unless the already increased pressure becomes suddenly and temporarily still greater, an event which of itself may be enough to bring on rupture of the diseased vessel. A sudden intensification of pressure, such as is meant, may arise from emotional causes, or from bodily exertion involving great muscular effort, from a full meal, from the use of cardiac stimulants, such as alcohol, coffee, and even digitalis (if given when arterial tension is already high, as Traube[1] has observed).

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[1] Med. Times and Gaz. 1855.

In the same category are to be reckoned the cases where persons are attacked with apoplexy in a cold bath (causing extensive contraction of the cutaneous arteries).

Up to this point we have spoken only of changes in the arterial system; how does the case stand, however, with increase in the venous pressure? Without doubt, this condition may act as an exciting cause of rupture, as the cases prove where the stroke has occurred under the influence of straining at stool, or from coughing, sneezing, or laughing. The mechanism of the process is clear; but, as in the case of the arterial pressure, so here also, disease of the vascular walls is probably invariably present. A sufficient proof of this seems to me to lie in the fact that, in whooping-cough, intra-cranial hemorrhage is almost unheard of, in spite of the great frequency of the disease, and the immense increase in venous pressure which attends the paroxysms; and this because the patients are at an age when changes in the vascular walls are but seldom met with. Similarly, this event is of the rarest occurrence as a consequence of labor, in proportion to the number of child-births; though here also, the venous stasis certainly reaches a notable intensity.

Equally rare is hemorrhage during epileptic attacks, though accompanied with the most intense cyanosis; this fact is admitted by Reynolds. On the other hand, it has been repeatedly observed in case of thrombosis of the sinuses. (Vide the appropriate chapters.)

Various affections, which seem to have in common some morbid *change in the composition of the blood,* appear exceptionally to lead to hemorrhage. Although, for the majority of these cases, the exact nature of the so-called alteration is totally unknown, it is, as a rule, assumed that, in consequence of insufficient nutrition, the vascular walls have become more brittle than normal. Without undertaking to deny this fact—about which there is but little that is positive to be said—we would yet call attention to the following points:

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[1] Berl. klin. Wochenschr. 1871. No. 38.

Intra-cerebral hemorrhages occur only exceptionally in connection with the pathological conditions in question. Although no statistics can be adduced to support the statement, still the estimate is surely a safe one, that out of thousands of cases of typhoid fever, for instance, cerebral hemorrhage is not met with more than perhaps once. Similar statements might be made with regard to pyæmia, icterus, and the so-called hemorrhagic diathesis.

Considering the rarity of occurrence of actual hemorrhage under these conditions, although the assumed alteration in the blood and in the walls of the vessels must be supposed to be present in all of them in some degree, and taking into consideration also instances like that recorded by Vulpian and Charcot, where the extravasation of a large amount of blood into the brain, which took place in the course of an attack of icterus (due to cancer of the liver), was found after death to be associated with the presence of miliary aneurisms, it seems fair to raise the doubt whether the blood diseases mentioned, to the list of which scurvy and puerperal fever may be added, are capable of causing cerebral hemorrhage, unless occurring in connection with further anatomical changes elsewhere. We repeat that we do not venture to give deliberate judgment in this matter, not regarding the proper time for doing so as having yet arrived. In the meantime we will add that Ollivier and Ranvier[1] have brought forward two original and six borrowed observations, to show that *leucocythæmia* may give rise to meningeal and intra-cerebral hemorrhages: they refer them to the stasis of the white blood-cells. The extravasations were partly capillary, partly of moderate severity.

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[1] Arch. de Physiol. Normale et Path. 1870. T. III.

Finally, it may be mentioned that the hemorrhages, observed now and then in the course of the pernicious anæmia recently described, especially by Biermer,[1] Gusserow,[2] and Immermann,[3] deserve to be placed in this category.

A number of different conditions bear the reputation of acting as *predisposing causes* of cerebral hemorrhage. The most important among these is *the age of the patient*. One fact in this condition is too evident to excite discussion: hemorrhage is rare before the fortieth year, very common after it. On the other hand, there are several matters of detail about which opinions differ.

Thus, it has been believed that apoplexy (for it is the occurrence of this symptom, and not of the hemorrhages, strictly speaking, that has been made the subject of statistical study) occurs the most frequently between fifty and seventy; after that, again, less often. Burrows[4] was the first to show that in this calculation the influence of the fact, that the number of persons living at an advanced age grows progressively less as their years increase, had not been taken into account at all. According to his reckoning, not only does the relative frequency with which hemorrhage occurs increase constantly from twenty to eighty years, but even the absolute number of cases increases up to seventy years, in spite of the relative smallness of the population at that age. The rarity of hemorrhage in youth is acknowledged; but yet it is not so rare but that it is occasionally met with, even in childhood, by every physician. I have myself observed several such cases. Sormani[5] has published two tables, one embracing 3,678, the other 9,653 cases, of which the latter is quite useless for our purposes, because it groups together all possible acute cerebral affections; indeed even the former speaks only of apoplexia fulminans in general. By this one, however, the statements made by Burrows are confirmed; further, the affection is found to be of least frequent occurrence between the ages of four and twenty-two, somewhat more common below four. This is no doubt to be explained by the relative commonness of meningeal apoplexy in early childhood.

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[1] Correspondenzbl. f. schweiz. Aertze. 1872. Jahrg. II.

[2] Arch. f. Gynäkol. 1871. Bd. II.

[3] Deutsch. Arch. f. klin. Med. 13. Bd.

[4] On Disorders of the Cerebr. Circulation, etc. London, 1846.

[5] Riv. Clin. 1871.

The direct relation between cerebral hemorrhage and the age of the patient, on the one hand, and between this latter condition and the presence of miliary aneurisms, on the other, is too clear to be questioned.

Although the statement of Joseph Frank, that women are attacked with apoplexy only in the proportion of one to ten, is greatly exaggerated, yet it is certainly, in general, true that men are more subject than women to cerebral hemorrhage ; perhaps because they are more often exposed to exciting causes.

As to the influence of *occupation*, there is not much to be said ; Sormani points out with reason that the fact that the average length of life of persons engaged in the different pursuits varies greatly, is to be taken into consideration in determining this point. As in the case of the cerebral hyperæmias, so also it is true of cerebral hemorrhage, that it occurs the least frequently in summer, the most frequently in winter. The explanation of this fact seems to us to be reached by Hermann's investigations on the effects of drinking cold water (Pflüger's Arch. 3. Bd.). The nature of the influence—if there be any—exerted by changes in the atmospheric pressure at the different parts of the day, by climate and by race, has not yet been satisfactorily determined by statistical evidence.

One factor remains to be considered, to which formerly much significance was attached, while latterly it has been almost absolutely disregarded, namely, the influence of the so-called apoplectic or plethoric habit, characterized, as is well known, by the conjunction of broad chest and shoulders and short neck, with large abdomen, powerful muscular system, and reddish face.

It is to be remarked, in the first place, that poorly-nourished, thin persons (patients of this type form, indeed, the majority among hospital cases) may be attacked with hemorrhage as well as those of the opposite type—that is, they are just as liable to have the real cause of the affection, the diseases of the arteries. It might, however, be questioned whether the most important exciting cause of the attack, the increased pressure upon the walls of the vessels, is more likely to be present in connection with the apoplectic habit. A definite opinion on this point cannot be given; in our own experience, anything which might be called an apoplectic habit was absent in the majority of cases. We are inclined to think that the popular fear of apoplexy, when the characteristics referred to are present, is based rather on the confounding of this affection with cerebral hyperæmia and its attendant symptoms. At the same time it cannot be denied that repeated hyperæmias are occasionally followed by hemorrhage, and, in so far, these popular fears are not without foundation.

The observation that cerebral hemorrhage is apt to occur in certain families favors the idea that such a tendency may be hereditary. Hughlings Jackson points out, with justice, that this tendency can be but an indirect one, making itself felt only through its tendency to promote the primary diseases in the arteries.

Pathological Anatomy.

As a rule, cases of cerebral hemorrhage are divided anatomically into two forms: those where the extravasated blood is collected into a mass, or so-called focus, and the capillary apoplexies. We will treat, first, of the *apoplectic foci*, strictly speaking.

These foci may be of every possible size, being at times as small as a lentil,[1] at times so large as to almost entirely destroy an entire hemisphere. The extremes, especially the latter, are met with but seldom. The commonest size is between that of a hazel-nut and that of a lady-apple (Kleinapfel). In such cases their form is more or less round; nevertheless hemorrhages are met with, especially in the pons Varolii, in the white substance of the brain, and in the neighborhood of larger foci, with which they are connected, where the blood is spread out to a greater or less extent in layers; indeed, they may take on the greatest possible variety of form. In the cortical substance, particularly, they are rather apt to spread out laterally.

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[1] About the size of a large pea.—TRANS.

The number of the foci is likewise apt to vary. As a rule, they occur singly; but two, four, or even more, may make their appearance simultaneously. In this connection it is worthy of notice that hemorrhages sometimes occur simultaneously in symmetrical parts of the brain, the two thalami optici or corpora striata, for example. It is not uncommon to find, in addition to the recent extravasation which has been the cause of death, the remains of older extravasations in the form of cysts and scars (vide below). This fact accords with the clinical observation that the same individual may be a victim of repeated seizures.

Daily experience teaches that the vascular ruptures occur much oftener in certain parts of the brain than in others. Their favorite seats are *the corpus striatum and the nucleus lenticularis*,[1] *with the neighboring parts of the hemispheres, and the thalami optici.* To such a degree is this the case that, according to Andral, out of 386 cases the hemorrhage was situated 61 times in the corpus striatum, 35 in the thalami optici, 27 in the centrum ovale of Vieussens, 202 in the above-mentioned ganglia, together with the neighboring brain substance. Rochoux also, out of 70 cases, found the extravasation 43 times in the corpus striatum, and 4 or 5 times in the thalami optici. It is for this reason, as Hasse very properly points out, that the vast majority of the cases of cerebral hemorrhage conform clinically to the same type. It is not possible to draw conclusions as to localization by tabulating the large numbers of cases reported in the literature of the subject, because, as a rule, only those cases have been recorded where the situation of the hemorrhage was peculiar, not those of the common form, where it occurred in the cerebral ganglia.

The reason for the disproportionate frequency with which the parts mentioned are attacked lies primarily in the anatomical relations of the arteries which ramify in them. According to Heubner and Duret (ll. cc.), the branches first given off from the anterior and middle cerebral arteries supply the large anterior end (the caput), and the middle portion, of the corpus striatum, together with the nucleus lenticularis, and, further, the capsula externa and interna. These two arteries are now, in virtue of being the immediate prolongations of the trunk of the carotid, exposed more than any others to the effects of sudden increase of the heart's action; in other words, direct and indirect causes unite to make the districts which they supply peculiarly liable to be the seat of hemorrhage.

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[1] Nucleus extraventric. corp. stria.—TRANS.

Next to these districts, in point of vulnerability, come, as we descend the scale, the various divisions of the cerebral lobes (exclusive of the cornu Ammonis), no difference existing between the lobes, in this respect, except that the occipital lobes are the least often affected; then, after a considerable interval, comes the cerebellum, and a little below it the pons, in conjunction with which the pedunculus cerebri is often attacked.

Isolated foci in the pedunculus are of as rare occurrence as they are in the medulla oblongata and the corpus quadrigem. They are almost never found in the cornu Ammonis, the corp. callosum, or the fornix; and there are other districts, as for instance the crura cerebelli, where they occur only very exceptionally.

Hemorrhage into the ventricles hardly ever occurs except when the blood breaks through from the neighboring parts; thus it may escape into the lateral ventricles from the corp. striata or thalami optici, and into the fourth, either from these same parts (after passing through the third), or, what is more common, from the pons. Rupture of the vessels of the ventricles themselves takes place but rarely.

Blood extravasated in the interior of the brain may make its way to the outer surface, either by breaking directly through the intervening substance, especially if that be the cortex alone, or by traversing the third and fourth ventricles. The outer surface then becomes covered with a layer of blood of varying extent and thickness.

In case a mass of blood of some size is extravasated in one hemisphere, the dura mater of that side is put on the stretch, and the falx pressed over towards the opposite side; the convolutions are somewhat flattened, and the sulci more or less completely obliterated. The pia and the uninjured parts of the affected hemisphere are often, under these circumstances, found anæmic.

The *apoplectic focus in its fresh condition* consists of a dark red clot, which is ordinarily uniform in character throughout.

Rokitansky[1] distinguishes between two groups of cases, in one of which the fibrin collects rather towards the centre, in the other rather towards the periphery of the clot, as corresponding to differences in the readiness with which repair takes place ; but a sharp line of division is not to be drawn between them.

The injured brain substance forms with the extravasated blood a sort of grumous mass. The wall of the focus consists, at the part nearest the clot, of torn shreds of cerebral tissue ; then follows a zone of varying thickness, averaging a few lines, made up of softened tissue, saturated with blood serum, and often the seat of capillary apoplexies ; within the central mass itself, close search will generally discover the remains of ruptured vessels, although for most of them the laceration was plainly an affair of secondary occurrence. If the plan is adopted of floating out the mass under water, which should be repeatedly poured off and again renewed, it will be found possible to pick out the very miliary aneurism from which the primary extravasation took place (Bouchard).

If the hemorrhage does not end fatally after a few hours or days, *structural changes begin to make their appearance, both in the blood-clot itself and in the surrounding brain substance.* The former becomes converted into a dark, chocolate-colored mass, of the consistency of gruel, the fluid constituents of which are soon reabsorbed. As a result of this latter process, the color of the mass grows lighter, becoming something between red and saffron yellow. The tissues forming the walls of the apoplectic focus often become softened by the imbibition of serum, as above said, to a marked degree, during the first few days. A retrograde, fatty metamorphosis of the torn fragments of brain tissue, analogous to that which occurs in the fibres of peripheral nerves cut off from their nutritive centres, plays its part in bringing about this softening. Finally, a third kind of change often occurs, consisting in an inflammation of the surrounding cerebral substance, which may at length become so extensive and cause such widespread œdema as to prove fatal.

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[1] Lehrbuch d. path. Anat. II. Bd.

In case the patient survives the attack for several weeks or months, the hemorrhagic focus becomes transformed into a sort of *cyst*. Virchow[1] pointed out, years ago, that these cysts are not simple cavities filled with fluid, but that they almost always contain in addition loose, spongy connective tissue. The fluid contents of this œdematous connective tissue vary in color from brown to bright yellow—the pigment, which owes its origin to the red blood corpuscles, being either granular or cystalline, and often enclosed in cells. Sometimes the pigment disappears altogether, leaving a cloudy, whitish, and whey-like fluid.

The wall of the cavity may be lined with firm connective tissue, either pigmented itself, or covered on the surface with pigmented material, in which case it is smooth, and shows no trace of the ragged appearance which it presented in its fresh state, or it may be formed by a layer of soft, friable tissue, containing granular bodies and detritus, which passes over immediately into the normal cerebral tissue (Foerster[2]).

These cysts—which vary in size between that of a peach-stone and that, as an extreme limit, of a hen's egg—may at times persist permanently without change. In many cases, however, the seat of the hemorrhage is found at a later date to be occupied, not by a cyst, but by the so-called *apoplectic cicatricial tissue;* and it is impossible to say with certainty whether this tissue is a primary formation, following directly on the state of things produced by the hemorrhage, or whether it is the result of a transformation of the cysts. This cicatricial tissue occupies a space of considerable superficial area, but of no great thickness, presents more or less flattened rust-colored surfaces, and is either made up throughout of simple, dense connective tissue, or of layers of such tissue, enclosing between them a layer of softer, friable or spongy substance.

In case the hemorrhage occupies a limited space in the cortex cerebri and the process of repair goes on properly, the same changes take place as where the more central regions are affected, with the single difference that, in the former case, the pia mater is involved. The cerebral substance is found, under these conditions, depressed, of yellow color, and adherent to the pia. At times there are found here also cavities containing loose connective tissue, its meshes filled by serum, which has evidently been secreted by the vessels of the pia.

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[1] Virch. Arch. I. Bd. p. 454.

[2] Pathologische Anatomie.

When hemorrhagic foci are of long standing they may give rise to various *secondary changes*, not only of the parts in their immediate neighborhood, but also of others in distant regions. These changes may be of either of two kinds: first, such as are limited in their action to certain tracts of conducting fibres, which have been severed at some point in their course by the extravasated blood; and, second, such as involve the entire mass of the brain. Although Cruveilhier noticed and called attention to the former variety of these secondary changes, yet the credit of having studied them thoroughly, and in some respects even exhaustively, unquestionably belongs to Türck;[1] and among the investigators who have followed in his steps, Bouchard deserves to be specially mentioned. According to the latter observer, the secondary degeneration of the motor nerve tracts fails to occur when the superficial layers of the cortex are the exclusive seat of the lesion; it takes place only when the deeper layers of the cortex and the white substance lying beneath are affected as well. It has been observed with especial regularity in the cases where the seat of the hemorrhage was in the corpus striatum, and in its greatest intensity where the capsula interna was involved (Charcot and Vulpian). It seems to be much less marked when the hemorrhage takes place into the thalami optici or centrum ovale. The effect of hemorrhages into other parts of the brain, as regards their tendency to produce these secondary changes, has not yet been studied with especial care.

This secondary degeneration, the histological characteristics of which we have not space to go into here in detail, is met with only after the lapse of several months, and consists essentially of an atrophy of the nerve-fibres and the development in their place of connective tissue. The path which the degeneration follows takes it, when the extravasation is situated in the localities mentioned above, through the crus cerebri, the pons, and the anterior pyramids. Within the latter the degenerative process passes over, in its descent, to the other side of the medulla, and may thence continue its course downwards through the entire length of the spinal cord, occupying especially the posterior portion of the lateral column. It may also affect the cord to some extent on the same side with the lesion, in which case it remains confined to the inner part of the anterior column.

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[1] Zeitschr. d. Gesellschaft der Wiener Aerzte. 1848 and 1852.

Degeneration of the peripheral motor nerves under these circumstances has not as yet been demonstrated; and, indeed, the fact that their excitability to electricity is preserved makes its occurrence in them appear improbable (vide below). Meissner[1] has reported two cases of apoplexy in which he found degenerative changes in the touch corpuscles of the skin. Langerhans,[2] on the other hand, examined seven such cases, four of which were of long standing, without being able to discover anything of the kind.

The second variety of secondary change, which is from time to time met with, consists in a *general atrophy of the brain*, affecting either that hemisphere alone which is the seat of the lesion or both hemispheres together. We would refer to this form only in passing, as the subject will be treated of at length in another place. It need only be mentioned that this atrophy is said to be especially common in connection with extravasation, even if of slight amount, situated in the cortex cerebri.

It was pointed out above that it was formerly the custom to speak of *capillary apoplexy* as something quite distinct from the usual form of apoplectic hemorrhage. This term was devised to cover those cases where a number of minute, isolated blood-points, of the size of a pin's head, or even smaller, are found. These blood-points occur in connection with pathological processes of widely different kinds; thus they are met with in parts which are the seat of softening, or in the neighborhood of the ordinary large apoplectic foci. They are sometimes found also unassociated with such processes; under these circumstances they are due to small extravasations into the lymph-sheaths of the blood-vessels, and by their presence here they may give rise to genuine hemorrhage of some severity. They are sometimes met with in considerable numbers in the cortex cerebri, as a consequence of thrombosis of the venous sinuses. The so-called capillary apoplexy, then, either occurs as a secondary process or else it plays a comparatively unimportant part in genuine hemorrhage, acting as an occasional predisposing cause of the attack. If minute extravasations, such as those in question, of the size of a millet-seed or even smaller, really do take place, not into the lymph-sheaths, but into the cerebral substance itself, they are probably rapidly reabsorbed. At any rate, in the course of the numerous experiments in which I have had occasion to make simple punctures into the brains of animals, I have never been able to find any traces, after the lapse of about fourteen days, of the small quantities of blood effused around the wound.

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[1] Beiträge zur Anat. u. Physiol. der Haut. Leipzig, 1853.

[2] Virchow's Arch. 45. Bd.

Experimental investigations bearing directly upon the subject of cerebral hemorrhage have not, so far as we know, been made. There are absolutely no experimental facts which teach us anything with regard to the etiology of the disease. The results of the physiological examination into the functions of the different regions of the brain can, it is true, help us to determine with increased accuracy the seat of the extravasations. It must not, however, be forgotten that, in view of the structural differences between the brain of man and that of the animals, which for certain regions is not inconsiderable, the conclusions drawn from physiological experiments are only to be applied within very narrow limits to pathological cases. We regard it as very important, in the present state of our knowledge, to use great caution in adopting the results obtained from experiments on animals as a guide in the localization of circumscribed diseases of the brain. At the same time, as may be said in passing, we regard it as still less admissible to draw conclusions from the effects of disease as to the normal physiological functions of the injured parts, or on the same grounds to attempt to throw discredit upon the significance of carefully conducted experiments. We shall return to this matter again below.

Other sets of experiments bearing upon one or another special point in the pathology of cerebral hemorrhage may of course be utilized in the study of these questions. Such are the experiments upon the increase of intra-cranial pressure, especially those—already referred to above—by Leyden, and one by F. Pagenstecher, who succeeded, by introducing bits of wax within the skull, into the space between the brain and the dura mater, in reproducing to some extent the mechanical conditions present in the case of extravasations of blood. Within certain limits the results of all the experiments upon the circulation of blood in the brain can be turned to account. Taken all together, however, the help that we get from physiology, except in regard to a few points, is at best but inconsiderable.

Symptomatology.

Different individual cases of cerebral hemorrhage are liable to vary so greatly in regard to their clinical history that they may even fail to resemble each other in any respect. Nevertheless, in spite of their striking differences, almost all cases unite in presenting two clinical features, so constant in their occurrence that we have a right to regard them as *essential;* these are the suddenness of onset of the initial attack, often marked by the presence of the typical apoplectic symptoms; and, following this, the evidences of local cerebral disease (in Griesinger's sense), lasting for a longer or shorter time, often to the very end of life, and consisting, as a rule, in paralysis of one half of the body. There is in fact only one other cerebral affection which is characterized in an equal degree by these phenomena, and this is one which it is, indeed, often difficult or impossible to distinguish clinically from hemorrhage itself, namely, softening from thrombosis or embolism.

Premonitory Symptoms.

It has long been the custom to describe certain premonitory symptoms of cerebral hemorrhage (molimina apoplectica) as phenomena of frequent occurrence. In order that these symptoms may be appreciated at their true value, we would preface our description of them with the following observations: Characteristic premonitions of hemorrhage, in the sense in which one speaks of the prodromal stage of the acute exanthematous diseases, are never met with; in many cases there are no premonitions at all. Although we do not attach much importance to the evidence of statistics as regards this point, yet daily experience teaches how common it is for persons who have never before betrayed a single cerebral symptom to be suddenly prostrated by an apoplectic attack. On the other hand, it certainly happens at times that definite symptoms are present for a long period before an attack, such as justly awaken the fear of its approach. In a restricted sense, these symptoms may fairly be called prodromata, although even then not invariably so.

In the first place, it may be that we have to do with general symptoms, preceding the hemorrhage by several years, which, strictly speaking, have nothing whatever to do with the apoplectic seizure itself, a proof of which lies in the fact that they are by no means always followed by any seizure after all. The symptoms are, as a rule, dependent upon disturbances of the intra-cranial circulation, commonly of the nature of hyperæmia. Many of the precursory special symptoms, shortly to be mentioned, are also to be referred to the same category; some of these latter, however, forming a second group of the so-called prodromata, are, without doubt, sometimes due to minute extravasations, and it is possible that one reason why the remains of these extravasations are not oftener found after death is that they occurred in the very same part of the brain which became afterwards the seat of the fatal hemorrhage by which its structure was destroyed, and the investigation of former processes within its limits made impossible. Cases where attacks of paresis and alterations of sensibility appear in parts of the body, which later become paralyzed, are perhaps of this order. Then there is a third group of cases, where the so-called prodromata—under these circumstances only preceding the attack by a few hours or days—owe their origin directly to the hemorrhage itself, which has begun slowly to take place, thus constituting one part of the symptoms of the attack itself. We shall have more to say on this point further on.

As we view the matter, the symptoms of hemorrhage can only make their appearance contemporaneously with the hemorrhage itself; and we have only then a right to speak of premonitory signs when the primary or exciting causes of vascular rupture are themselves attended by symptoms, a matter with regard to which no general rule can be laid down, as will have been gathered from what has been said concerning the etiology of the affection. It can never be justifiable, in view of these same facts, to regard symptoms of this class as certain forerunners of hemorrhage.

Bearing these considerations in mind, let us proceed to the enumeration of the different varieties of these so-called premonitory signs. They may be classified, in the first place, as local and general. The former correspond pretty nearly to the group of symptoms which we have described as occurring in the lighter varieties of cerebral hyperæmia. Thus the commonest among them are frequently recurring dizziness, headache, ringing in the ears, muscæ volitantes, alterations in the disposition, either in the direction of irritability and pettishness, or, on the other hand, of drowsiness and lethargy. Especially important is a loss of the power of speech, making its appearance suddenly, and again, after a few hours, disappearing, and unattended with paralysis of the tongue. It would be impossible to discuss in detail the great variety of possible phenomena of this class, which are enumerated (rather indiscriminately) by Jos. Frank. Thus one patient is reported as having had, frequently, sensations as of delicious perfumes; another was constantly inclined to gape, etc.

The more strictly local molimina may also appear under various forms, and are met with, as a rule, though not invariably, in those parts of the body which are later to be attacked with paralysis. Thus some patients complain of a sense of weight and awkwardness, a slight degree of paresis in one arm, a leg, or in arm and leg at once, which, after a while, again disappears. Much more rarely, a certain heaviness of the tongue is seen, or unilateral facial paralysis, or even double vision, dependent upon the paresis of one of the muscles of the eye. Side by side with these motor disturbances, or independently of them, the patients complain of abnormal sensations in the extremities, among the most common of which are a sense of stiffness, a feeling of "pins and needles," formication, or sudden attacks of sensations of heat and cold, or of pain of an indescribable character. Not unfrequently disturbances of vision are met with as prodromata. Apart from the diplopia above referred to, experience has shown that diseases of the retina, especially those which accompany chronic nephritis, usually underlie these visual disturbances.

In a few cases complete amaurosis has been observed to precede attacks of apoplexy by several days. E. Berthold[1] discovered in one case a unilateral hemorrhage into the retina, as cause of a difficulty in vision of a few days' standing; and later the patient died from an attack of apoplexy under his immediate observation; unfortunately no autopsy could be made. Exceptionally it happens that nose-bleed occurs as a premonitory sign.

All these premonitions precede the final attack often by weeks or months, sometimes by years. We would refer again to the similarity between this group of symptoms and those which accompany simple cerebral hyperæmia; at the same time we would repeat that it is often entirely wanting, and refer to the explanation given above as to its origin.

The First Stage of the Hemorrhage.

The beginning of the intra-cerebral hemorrhage, whether preceded by premonitions or not, may be marked by any of a variety of symptoms. As a rule, the occurrence of the extravasation is accompanied by loss of consciousness; but this is not invariably the case. As was remarked at the outset, the sudden loss of consciousness is designated as apoplexy, or the apoplectic shock; hemorrhage may then occur, either with or without apoplexy. The former occurrence is by far the more common; but, in either case, manifold differences in the symptoms present themselves in different cases. We shall first discuss the apoplectic hemorrhages.

In very rare instances, though they do occur, the classical state of things, answering to the name which designates it, is observed. In the midst of apparent health, while engaged in some indifferent occupation, the patient utters a cry, and falls directly, sometimes even without so much warning as this, as if struck by lightning, to the ground. The mass of the extravasated blood under these circumstances is not always very large; it may indeed be small. On the other hand, its seat is almost always in about the same region, namely, in the pons, the medulla oblongata, or the cerebellum—in saying which we do not mean to imply that the attack is always of this form when the seat of the lesion is in one of these parts.

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[1] Berl. kl. Wochenschr. 1869. No. 39.

Far oftener the apoplectic attack has a more gradual beginning. The patient complains of dizziness, of abnormal sensations, or of pain in the head; or he complains of some of the symptoms referred to above among the prodromata; or his mind becomes confused, and his speech inconsequent; or, while his consciousness may be apparently undisturbed, he can no longer express himself in words; or he becomes sleepy; or nausea, and even vomiting make their appearance, accompanied with a sense of great exhaustion, muscular relaxation, constantly recurring attacks of chilliness. After this condition has endured for some hours (exceptionally even days), during which time perhaps venesection has been practised, the stage of unconsciousness comes on.

Again, in still other cases, one of the extremities, the arm, the leg, or some of the muscles of one side of the face or of one eye, becomes the seat, not only of a simple sense of weakness or a partial loss of power, but of complete paralysis. Occasionally the scene opens with slight clonic convulsions, or even with tonic spasms, in certain groups of muscles, which later become paralyzed; and not until after these symptoms—perhaps even hours after—does the patient fall into a state of sopor.

Not less varied than the character of the attack at its commencement are the special symptoms which are met with after loss of consciousness has set in. As a rule (and in this consists the fundamental peculiarity of the condition), the victim of the seizure lies plunged in the deepest sopor.[1] Consciousness is completely extinguished—no trace of voluntary movement, no sign of any kind, betrays the least glimmering of conscious perception. In the severest cases even the reflex motions are abolished (the extremities losing all tendency to assume a definite position, which in health never happens, even during sleep), and in its place we find a universal, corpse-like relaxation of the muscles; only the movements of respiration and of the heart, together with the power of swallowing, which is seldom absent—provided that the fluid is once brought fairly into the pharynx—remain to indicate that life persists.

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[1] The older physicians used to distinguish three varieties of sopor, corresponding to the relative intensity of the symptoms—namely, coma, lethargus, carus. The sopor from apoplectic hemorrhage should therefore properly be called carus.

It is not in every case, however, that the shock is so profound as this: sometimes powerful irritations, such as usually cause pain, may be able to call out unconscious reflex movements. In these cases a careful examination can discover a difference between the two halves of the body—the extremities of one side offering a certain resistance to passive motion, while those of the other sink, when unsupported, like a lifeless mass; likewise the corner of the mouth on one side stands somewhat lower than that of the other, and the opposite naso-labial fold is very strongly marked. In short, we have to do here with a case of hemiplegia, of unilateral paralysis, which has made its appearance at the same moment with the sopor.

In case the general muscular relaxation is so great that the hemiplegia is not to be recognized, and that for this reason doubt exists as to whether the apoplexy is due to a cerebral lesion, especially hemorrhage, or to some other cause (opium poisoning, asphyxia, etc.), it is important to make search, according to Prevost, for one symptom, whose presence generally (according to Prevost always) indicates that we have to do with a demonstrable affection of the brain; this is the deviation of both eyes towards the non-paralyzed side, a condition which is often associated with a rotation of the head in the same direction.

This phenomenon—which is not to be confounded with unilateral strabismus—is observed especially in sudden attacks, and is of short duration, lasting usually only a few days.

According to Prevost, it is found with greatest frequency when the lesion is seated in the corpus striatum and its neighborhood, but also at times with lesions of other parts, and is to be classed in the same category with the so-called "compelled movements" (Zwangsbewegungen) so often observed in the course of experiments upon animals.

I am able to confirm this statement in the main, but with the restriction that I have seen this same phenomenon in cases where no anatomical lesion existed (vide, for instance, a case reported above as due to cerebral hyperæmia). Eulenburg has also pointed out that it may occur in connection with unilateral epileptic attacks.

Sometimes the onset of the shock is attended with *convulsions.* This happens but rarely, and only under certain definite conditions, namely, in cases where the amount of extravasation, which then generally has its seat in the hemisphere, is enormously large, and in those where it is seated in the pons and medulla oblongata; still more if it has also broken through the floor of the fourth ventricle. In the former case the convulsive action is generally very slight, only feebly suggested; in the latter it may be very severe, closely resembling the convulsions of epilepsy: in both cases it is bilateral. Hirtz endeavors to show that the convulsions are much more common when the hemorrhage breaks through into the ventricles (fifty-three times out of seventy-seven cases); but this occurrence probably has no other physiological significance than that attaching to these cases in common with all extensive hemorrhages. It happens only very exceptionally that, during the coma following hemorrhage, convulsive action occurs, which is limited to the paralyzed limbs. On the other hand, it frequently happens that the extremities which are afterwards to be paralyzed are found at first in a condition of tonic spasm. This is said to occur with special frequency when the hemorrhage has destroyed the walls of the lateral ventricles; but this statement is in need of further proof. Durand-Fardel, who has been its special advocate, brings forward a number of instances, on his side; yet certainly the rule is by no means an invariable one.

The other features of the attack, the appearance of the face, the condition of the pupils, the respiration, the pulse, are also subject to manifold variations. The color of the face is sometimes deep red, even slightly cyanotic, the latter indicating an irregular and impeded respiration. At other times it is entirely normal, and again in other cases it is found to be quite pale, resembling in every particular that of a person in a faint. This last appearance seems to occur with especial frequency in cases where the hemorrhage has taken place gradually. Sometimes in the very beginning of the attack, before consciousness is lost, the patients look pale, with sunken features, and they then, as a rule, suffer from nausea and vomiting. On this generally follows sopor and the venous congestion of the face. The state of the pupils (with regard to this point we agree with Hughlings Jackson) is as little characteristic as the appearance of the face; they are sometimes dilated, sometimes also of normal size, or, again, unusually contracted. A high degree of myosis is an almost invariable symptom in case of hemorrhage into the pons. Much more important is a marked difference in point of size between the pupils of the two sides (supposing the inequality not to be a physiological peculiarity of the individual), because it points to a unilateral affection of the brain. The character of the pulse also varies with the case; the so-called pulsus cerebralis of the older physicians, *i. e.*, a slow, sometimes also irregular pulse, is, to be sure, the variety most often observed; but sometimes, on the contrary, it is very rapid and entirely regular; the tension is equally subject to variation. The *respiration* is in many cases quiet and as regular as that of a person in sleep. Our fathers used to describe a noisy, snoring, "stertorous" respiration as peculiar to hemorrhagic apoplexy; but, as just stated, it is not universally present, and is, moreover, met with in sopor due to other causes. In case of very deep unconsciousness the cheeks are drawn in during inspiration and puffed out during expiration (the so-called tobacco-smoker's respiration). Traube[1] was the first to call attention distinctly to the phenomena of the Cheyne-Stokes respiration in cerebral hemorrhage, and we are able to confirm his statements. If the patients do not awake from their coma, the respiration becomes, towards the end, intermittent and irregular. Finally, it is to be noted that the fæces and urine sometimes pass away involuntarily during the sopor.

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[1] Berl. klin. Wochenschr. 1869. No. 27.

Such is the description of the true apoplectic attack. At the same time the old notion, that the sopor due to cerebral hemorrhage is always of the profoundest kind, has long since, by common experience, been disproved.

In fact, it happens not unfrequently that such patients become only somnolent and answer when aroused by calling, though they may be at the same time confused in mind. These latter forms bridge over the interval between that last described and the one to which we shall next direct our attention, namely, where hemorrhage occurs unattended by apoplexy—a condition which, though comparatively rare, is unquestionably met with from time to time. In our belief the frequency with which these cases are to be seen has been underrated. We have no statistical evidence to offer, it is true, but we are well convinced that cases are often called apoplectic by the laity where a paralytic attack comes on suddenly, even if unattended with coma, and that their faulty impressions are then transmitted to the physician; certainly the proportion of cases must be small which the physician has the opportunity of observing in person from the very outset.

It sometimes happens that the hemorrhage, with all its consequences, occurs without the patient's having lost consciousness even for a single minute. It is self-evident that under these circumstances all those phenomena will be absent which belong to the apoplectic seizure as such; while, nevertheless, the whole group of remaining symptoms, which are the result of the extravasation regarded as a localized disease, may make their appearance in due course. Among these symptoms are to be reckoned all the various molimina prodromalia, the manifold phenomena that immediately precede the attack, and, finally, those which remain permanently after the return of consciousness in the usual cases. Such patients notice that they can no longer use the arm properly at their work, and that it feels a little stiff; the same thing is found to be true of the leg of the same side; these extremities may next become the seat of slight muscular twitchings; the weakness increases; the patient falls to the ground, is picked up and laid upon a bed, and after half an hour, or an hour, from the beginning of the attack the paralysis of one side of the body is complete. All this time there may have been not the least disturbance of the sensorium.

In the cases of this kind that we have ourselves observed, as well as in the greater part of those recorded in the literature of the subject, the paralysis occurred rather gradually; now and then, however, it develops with great rapidity. Thus Trousseau relates the case of a woman, who, while at table with her family, and feeling herself in perfect health, noticed that she could no longer cut her bread ; wishing to express her astonishment at this, she found that her articulation had become indistinct; and on trying to rise from her chair, she fell to the ground, from paralysis of one leg; yet her consciousness had not been lost for a single moment. Andral gives a similar instance, with autopsy.

Before we go further in our study of the symptomatology, it may be well if we stop for a moment to consider the question, What are the influences that give rise to the apoplectic attack? We shall confine ourselves, in the following discussion of this point, exclusively to the conditions which are present in cases of cerebral hemorrhage. Unquestionably there are several different factors which, at the moment of occurrence of the extravasation, conduce to bring about loss of consciousness; it is certainly impossible to refer it always to the same cause. We are driven to this conclusion by a simple consideration of the following facts : 1. That the symptoms of apoplexy may attend small as well as large extravasations. 2. That, in spite of the fact that they occur in consequence of some of the smaller hemorrhages, they may fail to attend the larger. 3. That rapidly occurring extravasations do not always cause loss of consciousness, while, on the contrary, this result does sometimes follow those whose development has been gradual.

At the head of the list of determining influences, because most readily studied, we shall place the *sudden increase of the cerebral pressure* which is exercised by large extravasations, and which, as has been proved experimentally by Leyden and Pagenstecher, regularly gives rise to coma. This condition is without doubt the cause of the apoplexy in those cases where one hemisphere becomes the seat of an extravasation of such a size as to push the falx cerebri to one side, or where the blood breaks through into the ventricles, and finally makes its way even to the surface of the brain. Abercrombie, in his excellent treatise on the Apoplectic Shock, gives it as his opinion that the essential factor in the production of the symptoms in cases of this kind is *not the direct compression of the nervous elements*, but the disturbances of the circulation due to the mechanical action of the extravasation; and the nature of these disturbances of circulation is now universally regarded to be anæmic. If the statements which we made in speaking of the pathology of cerebral anæmia, as to the varying readiness with which the different parts of the brain respond to circulatory changes, are borne in mind, it will appear comprehensible why it is that general convulsions so rarely arise in connection with coma hemorrhagicum, namely, because extensive hemorrhages are, on the whole, of infrequent occurrence.

Without feeling obliged to admit the force of the calculations made by Pagenstecher, as a corollary from his experiments upon the effect of the injection of wax into the skull cavity of dogs, to the effect that a pressure, amounting on an average to 40 grms., in maximal cases to 90 grms., ought to be borne by the human brain without causing symptoms due to the pressure itself, yet we may admit as certain that in some cases *extravasations, whose amount is much less than that of the cerebral fluid,*[1] may give rise to apoplexy. According to what was said in the introduction (on Intra-Cranial Circulation), this fluid must first be expelled before the effects of the pressure can be felt by the brain itself, *i. e.*, before anæmia from mechanical expulsion of the blood can be brought about. In all probability the amount of the extravasated blood must be even greater than this before it can compress the vessels even in part. Experience, however, teaches that very extensive hemorrhages occur only exceptionally. We find ourselves thus driven to the conclusion, that certainly in the case of the small extravasations, probably indeed in that of the majority of all cases, some other influence besides the increase of cerebral pressure, and the consequent anæmia, is to be regarded as the cause of the apoplexy.

What, then, is the nature of this influence? Possibly in certain cases a hyperæmia of some intensity, which, while tending to favor the vascular rupture itself, helps also to call out the symptoms of coma. That this supposition is an admissible one, is made evident by a study of the symptomatology of cerebral hyperæmia, and it is in every way possible that the influence referred to really makes itself felt in certain cases. These cases are, to be sure, probably exceptions to the rule; for it has been shown above that it is only in the minority of instances that any degree of congestion worth mentioning is present in connection with the apoplectic shock. We have not, then, even yet discovered an explanation sufficient to cover every case.

Trousseau, in his Lectures, assumes the existence of a sort of temporary stupefaction of the brain (étonnement cérébral), supposed to be induced by the hemorrhage, to explain the occurrence of the apoplexy; while Hughlings Jackson refers the same to the effect of "shock," and Jaccoud to "névrolysie." The condition designated by these terms is believed to be brought about by the hemorrhage, in virtue of the suddenness of its occurrence, as in the analogous case, where a similar condition is excited by injuries. The authors referred to give, it is true, no definite statement as to exactly what they understand by this "brain shock," nevertheless this view has unquestionably its attractive side; it would furnish an explanation of the fact that even very small extravasations may give rise immediately to coma. A condition, analogous to that of this "brain shock,"[1] may be produced experimentally on animals by section of the spinal cord, inasmuch as, *immediately* after this operation, the reflex excitability of the lower portion of the spinal axis is found to have been abolished. As for the case where the apoplectic symptoms are wanting, it might be assumed that the hemorrhage takes place so gradually as not to call them out.

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[1] According to Magendie, the quantity of the cerebro-spinal fluid amounts in the adult to an average of 62 grms., of which one-third must be allotted to the brain. Among old persons, in whom hemorrhage is relatively so frequent, the quantity is often much greater than this, having been found to amount, in cases of cerebral atrophy, even to 372 grms.

Although it is not possible either to prove or disprove this theory directly, yet the following arguments against it ought certainly to be brought forward. In the first place—and Trousseau himself has published examples of the kind—hemorrhage may take place rapidly and suddenly without causing apoplexy. In the next place, it might be argued that, in spite of the fact that some time elapses before the extravasation has entirely ceased, yet the commencement of the process at the moment of the breaking of the aneurism, must, in a certain sense, always be sudden, and ought, therefore, according to the theory in question, always to give rise to the symptoms of "brain-shock," which is not in fact the case.

Finally—and this is the main argument—the results of experiments upon animals are unfavorable to this theory: for certainly the sudden setting free of a mass of blood, or chromic acid, in the brain of rabbits or dogs, ought to excite at least some slight indication of this "shock;" yet in the course of the hundreds of experiments in which I have done this, I have never met with anything of the kind. It might, further, be supposed that the anatomical position of the extravasations would be of significance in determining whether apoplexy should result; an examination of reported cases gives, however, no support to this idea. Hemorrhages into the thalami optici, into the corpora striata, or the cerebral lobes, may alike take place without necessarily entailing loss of consciousness as a result.

Heubner[2] has very recently attempted to give still another explanation of apoplexy from hemorrhage. He points out that when an extravasation of any size takes place from one of the many arteries of the cerebrum (or from one of their branches, as, for instance, in the nucleus lenticularis), it must cause "a temporary sinking of the level in the reservoir which supplies it, represented by the arterial network of the pia mater." Owing, now, to the numerous anastomoses between the vessels of the pia, this change of pressure must, he supposes, make itself felt over the entire hemisphere. As a result of this, it is natural, as he considers, that symptoms of temporary disturbances of function of the cortical substance should make their appearance, such as syncope, loss of consciousness, and even apoplexy. It need only be said, as an argument against the universal applicability of this explanation, that loss of consciousness sometimes accompanies the extravasation of very small quantities of blood, such as would certainly be unable to cause any material change of level in the "reservoir of the arterial network in the pia," and also occurs in connection with hemorrhages such as are in no position to influence the circulation in the pia in any way, as, for example, those into the cerebellum.

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[1] A shock in the surgical sense—vide the chapter on Cerebral Anæmia—is hardly to be thought of as possible.

[2] Die luetische Erkrankung d. Hirnarterien. Leipzig, 1874. Note to p. 194.

We find ourselves then, finally, obliged to admit that the physiological relations of hemorrhagic apoplexy have not yet been made so clear as is commonly believed; and it is for this very reason that we judged it advisable to examine them at this length.

Let us now return to our study of the clinical history of the apoplectic attack. Sometimes, though certainly only in a minority of cases, the patient expires without having regained his consciousness. The length of time which elapses before the occurrence of this event varies for different cases. It happens only *very rarely indeed* that death supervenes immediately, within a few minutes. The shortest recorded period is five minutes (Abercrombie); as a rule, it amounts to several hours (from two to twelve) at least, and yet these cases are designated as apoplexia fulminans seu attonita; most frequently the sopor lasts for from one to three days before it is terminated by death. The characteristic symptoms of apoplexia fulminans are especially common in cases of hemorrhage into the pons Varolii and medulla oblongata, and their neighborhood, supposing that they are in such a position that the nucleus of the vagus nerve is subjected to pressure; but hemorrhages elsewhere, provided only their amount is sufficiently great, may lead to the same result, and this is also true of several different hemorrhages occurring simultaneously, and possibly of the coincident occurrence of intense hyperæmia with hemorrhage; pre-eminently, however, of that of meningeal and intra-cerebral hemorrhage. Death takes place, usually, in connection with symptoms which point to paralysis by pressure of the spinal centre of the vagus nerve (irregular faltering respiration, intermission and weakness of the pulse); or, in case the coma is of longer duration, also in consequence of œdema of the lungs or of pneumonia. It has been practically found that recovery rarely takes place after the coma has lasted for forty-eight hours.

Bourneville has studied with great care the behavior of the tempreature in apoplexy from cerebral hemorrhage. He finds that the temperature of the body at large is at first lowered (reaching 35.8° Centigrade = 96.5° F.), and remains so, in the fulminating form of the affection, until death. If life endures for from ten to twenty-four hours, the initial sinking of the temperature gives place to a marked and rapid rise. If death is to be postponed for several days, however, the initial period of sinking is followed by a stationary period, during which the temperature varies between 37.5° and 38.0° (99.8° F. and 100.4° F.), and finally, just before death, by a period of great elevation of temperature—a very unfavorable symptom.

Brown-Séquard[1] also has observed that, in case of animals, death, consequent upon injury to the brain, is often brought about by pneumonia. He discovered that when the crura cerebri and cerebelli, or the pons, are the parts injured, minute hemorrhages, or, on the contrary, anæmic spots, make their appearance in the substance of the lungs; after injuries of the medulla oblongata, œdema of the lungs was especially apt to occur.

In favorable cases consciousness begins to return after a longer or shorter period. In rare instances it comes back quickly, after a few minutes, as after a momentary faintness; on the average it remains absent from half an hour to three hours. The first sign of improvement, appearing while the sopor still continues, consists in the return of reflex excitability, exhibiting itself in response to irritation of the skin; consciousness next becomes gradually restored; but it rarely, even in the lightest cases, returns all at once to its normal state. Even when the restoration is relatively rapid, headache, and a sense of general discomfort usually remain behind. If the apoplectic symptoms have been at all severe, hours and even days go by before the direct consequences of the attack itself have disappeared, and the symptoms due only to the localized disease as such have become fairly developed. The patient is still inclined to be drowsy; his thoughts are confused; he gives sometimes inappropriate answers; is surly or apathetic; the movements even of the muscles which are not paralyzed are nevertheless feeble. Very often there is a certain awkwardness in the use of the tongue, and some degree of aphasia, which disappears after a few days, leaving no trace of its presence. In a few cases, certain mental disturbances, of the nature of delirium, are present for some days.

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[1] Comptes Rendus de la Soc. de Biologie, 1870, 5. Sér. T. II. p. 116; and 1873, T. III. p. 101. Lancet, 1871, Vol. I. p. 6.

We will for the present discontinue the discussion of the symptoms of the apoplectic attack itself, to return again to the subject at another time, and turn our attention instead to the further history of those cases where, as usually happens, the immediate consequences of the attack have, after a few hours or days, passed away, leaving only such disturbances behind as are dependent upon the permanent destruction of substance in the brain. At the first glance these disturbances appear, as a rule, strikingly uniform in character; but, on closer inspection, marked and varying differences are seen to exist between them.

It may be remarked, to begin with, that a case of cerebral hemorrhage often passes at once, without going through any intermediate stage, into this, so to speak, *chronic* condition, yet by no means always. It happens, namely, very frequently that symptoms make their appearance which are universally, and probably with justice, laid to the score of an inflammatory reaction, which is supposed to take place in the tissues surrounding the blood-clot. The intensity and therewith the attending symptoms of this inflammation are subject to variation. Supposing the patient to be restored to consciousness, and the sensorium to be quite or nearly free, he becomes under these circumstances confused anew, complains of headache or is restless, and shows signs of delirium. The bodily temperature rises from a few tenths of a degree to two degrees; at the same time other feverish symptoms appear, such as loss of appetite and thirst; the pulse does not always become more rapid—on the contrary, often slower; convulsive movements, even tonic contractions, come on in the paralyzed muscles, and the patient complains of more or less pain in these parts. These phenomena begin, as a rule, between the second and the fourth day after the attack, and are rarely of more than a few days' duration. The statement of Calmeil, that the convalescence from this condition, the return of appetite and of quiet sleep, does not begin until after two or three months, is correct only for individual cases; and even then the symptoms referred to are not constantly present, but are rather periodic, and usually of feeble intensity. The most constant feature of the affection is apt to be pain, while the symptoms of fever are much less strongly pronounced. These exacerbations may come on at intervals of two, four, or eight days. If the patient has passed over this period in safety, as generally happens, the chronic stage begins, and now for the first time it is possible to recognize, as Hasse fittingly points out, the amount of the mischief which has been done.

Permanent Symptoms. Motor Paralysis.

In this stage one symptom stands out in relief above all the rest, viz., paralysis of motion.

I regard it as unquestionable that the cause of this symptom is to be sought, at first mainly, later exclusively, in the fact that the integrity of the conducting tracts is actually destroyed through the mechanical action of the extravasated blood, and the transmission of motor impulses thereby rendered impossible.

This local destruction of the nerve tracts may act as a cause of paralysis, not only when it takes place in the pons Varolii or between that and the corpus striatum, but also when it occurs in the hemispheres themselves.

This statement is in contradiction to the earlier physiological theories, which declared it to be impossible that the paralyses accompanying—as it could not be questioned that they did—lesions in the hemispheres, should arise in the manner indicated.

Recent investigations (by Fritsch and Hitzig, myself, Ferrier) have shown that paralysis may occur as a result of lesions not only of the white substance, but also of certain definite regions of the surface of the brain.

Schiff[1] certainly goes too far when he says: "Even in cases of apoplexy the cause of the particular symptoms is not to be found by investigating the anatomical relations of the blood-clot found after death, and the extravasation is, perhaps, to be regarded rather as an accessory effect of the real disease, but in itself without significance for the production of the clinical result." The incompleteness of our present acquaintance with the symptoms of localized diseases of the brain can at most indicate how insufficient our present anatomical investigations frequently are in point of detail, but do not justify such statements as this.

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[1] loc. cit. p. 364.

Besides that just mentioned, there are still other modes in which extravasations of blood may under certain conditions cause paralysis. If the blood-clot is of considerable size, it will exercise such a pressure as to cause capillary *anæmia* in the neighborhood, and paralysis as a secondary consequence. The general relaxation of all the extremities that is met with in connection with large extravasations is certainly due to a widespread anæmia of this kind.

Further, the compression, stretching, or dislocation to which the nerve fibres or ganglion cells in the immediate neighborhood of the blood-clot are subjected probably also interferes temporarily with the function of those parts. The influence of these injuries, as well as that of the anæmia, is felt, of course, only at the outset of the affection, as it is soon counteracted by compensatory changes.

On the other hand, still another influence makes itself felt, which remains as an important cause of paralysis through the early part of the chronic stage of the affection, viz., œdema in the neighborhood of the extravasation, together with the capillary anæmia which it produces. This œdema is, in part, a result of the reactive inflammation which takes place, in part a result of the saturation of the tissues with serum from the exuded blood.

We need not discuss in detail in what manner these different agencies act in determining the *distribution* (at different stages of the affection) *and the duration of the paralytic symptoms.* We know from experience that, in the great majority of cases, the paralysis, during the first days after the occurrence of the hemorrhage, is much more widespread than at a later period. Certain of the muscles which were at first involved begin very soon again to obey the commands of the will; then follows a period of weeks or months, in the course of which a gradual improvement goes on in still other groups of muscles, ending in complete, or nearly complete recovery. After this no further improvement takes place, certain parts remaining in a paralyzed state during the rest of life, that is, for a period of ten, twenty, or more years.

As a rule, it happens (supposing the lesion to be seated, as is usually the case, in the motor cerebral ganglia, the nucleus lenticularis, the corpus striatum, and the neighboring parts) that the affections of the speech—whether consisting in complete loss of that faculty, or in simple awkwardness in the use of the tongue—or the derangements in the action of the ocular muscles, disappear before any other of the paralytic symptoms; next to these, control is gradually gained, to some extent, over the muscles of the leg and the face, the arm still remaining, however, more or less absolutely paralyzed.

It is especially worthy of notice that the paralysis of the leg almost always disappears much sooner and more completely than that of the arm, the reverse happening only exceptionally. A satisfactory explanation of this fact has not yet been offered. In our opinion more is to be said in favor of the following view than of any other: repeated experiments upon animals have proved that those motions that take place in symmetrical groups of muscles and bear the stamp of co-ordinated or associated movements—for example, swimming, springing, and the like—may be provoked by external excitations even after removal of the cerebral hemispheres—in other words, independently of voluntary innervation. There must therefore be centres for these motions, below as well as above the level of the corpus striatum (nucleus lenticularis). The motions of the lower extremities in walking are, in part, of the nature of associated movements, and the processes of innervation which underlie them are much simpler than those which produce the very complicated movements of the fingers and hand.

Such being the case, it seems not impossible that the voluntary effort necessary to the movements of walking, although prevented, in so far as it originates in the cortex cerebri of the affected half of the brain, from reaching the paralyzed side of the body, since the conducting tracts have been severed, yet might be able, starting from the cortex of the uninjured half, and acting upon these co-ordinating centres, to excite associated movements, which would involve the paralyzed side of the body together with the other side.

This view would be in harmony with the observed fact that in these cases of partial hemiplegia the associated movements of walking are more easily performed than isolated voluntary movements of the affected leg. According to the observation of Trousseau, the prognosis in cases of hemiplegia is much less favorable when the voluntary power over the arm returns soon, while that of the leg remains long incomplete or entirely absent. He believes that under these circumstances an early mental decay is to be expected.

The progress of the case is, however, not always that which has been indicated. Sometimes a lasting difficulty in speech remains; the movements of one-half the face may be permanently restricted, and for years the leg may continue partially paralyzed. On the other hand, in exceptional instances, all the paralytic symptoms may finally disappear. It is clear that these differences in clinical history are brought about by variations in the processes of regeneration in and about the focus of disease, and that they have to do also with peculiarities in the position of the latter. The function of the sphincters almost always remains normal, even though the extremities may be permanently paralyzed.

Respecting the *intensity of the paralysis*, it is plain, from what has been said, that it may vary between the slightest degree of feebleness in motion on the one hand, and absolute loss of motion on the other. In the nomenclature of the present day (which, it may be said, differs from that of the older physicians), incomplete paralysis is called "paresis;" the complete or nearly complete simply "paralysis." As has been said, in the early stages of cerebral hemorrhage paralysis is almost always present, but gives place gradually, in certain parts, to paresis. Occasionally, however, this latter condition is present from the outset, there having been no absolute loss of motion at any time. We shall speak below of the manifold further phenomena which are met with associated with the paralysis in affected limbs, but prefer to turn our attention now to the discussion *of the varying* *distribution* and characteristics of the paralysis in different cases (having reference always to the chronic stage of the affection into which it enters a few weeks after its commencement).

With few exceptions, this paralysis is *unilateral, and affects the side of the body opposite to that in which the lesion on the brain occurred;* this is the type of the so-called *cerebral hemiplegia.* Since the hemorrhage in the majority of cases is situated in the corpus striatum, the paralytic symptoms usually met with present the same, or nearly the same, features in every case; the arm and leg, the muscles of the face on the side opposite to that of the lesion, and not unfrequently those of the trunk, are paralytic or paretic.[1]

With regard to the paralysis in the extremities, as usually met with, we have only to add to what has been said, that when it is incomplete the flexor muscles *seem* to be more severely affected than the extensors (whether this is really the case, or whether it only appears so from the fact that the intensity of the paralysis in the two groups of muscles being equal, the position taken by the hand is only a reproduction of its normal position, is questionable; the latter view seems to us the more probable). I desire to emphasize particularly the fact that the muscles of the trunk are involved, because it is usually stated that they remain unaffected. If any one takes the trouble to examine a number of these cases with care, he will certainly observe that often during quiet respiration, still more markedly during forced respiration, the form of the chest on the paralyzed side (its antero-posterior diameter in the upper part and its transverse in the lower) changes less than that on the other side—those cases where any affection of the lungs or pleura is present being of course excluded. This difference is due to a paresis of the auxiliary muscles of respiration, as can be readily discovered by examination of the scaleni.

The weakness of the intercostal muscles, as well as of those of the abdomen and back, is much more difficult to demonstrate, and it is perhaps often wanting. A statement made by O. Berger[2] harmonizes with the results of our own observation, viz., that in many cases of hemiplegia from cerebral hemorrhage there is a greater or less degree of paresis of the trapezius and the levator anguli scapulæ on the same side with the paralyzed extremities (vide below, lesions in corpus striatum). Besides these muscles and those of the extremities, the muscles of the face are also paralyzed. It is a characteristic feature of this paralysis, as Todd was the first to remark, that those branches of the facial nerve which supply the frontalis, corrugator supercilii, and orbicularis palpebrarum, almost always escape intact, in which respect the intra-cerebral facial paralysis differs from the peripheral form, due to affection of the trunk of the facial nerve; in cases where the lesion is situated in certain parts of the brain, these fibres are, however, affected as well as the rest. Besides restricting the motions of the principal muscles of expression, and causing the drooping of the mouth on the affected side, the obliteration of the naso-labial fold, inability to whistle, to purse the lips, etc., the facial paralysis also interferes with the enunciation of the labials, and may even, as pointed out by Wachsmuth, impair the mobility of the tongue, by interfering with the action of the digastric and stylohyoid muscles. It may be due, in part, to the functional impairment of these muscles that the tongue deviates (with its point towards the paralyzed side), when thrust out; it is true that in some cases, as Eulenburg has shown, the deviation of the tongue in simple facial paralysis, without associated paralysis of the hypoglossal nerve, may exist only in appearance, due to the fact that it approaches the corner of the mouth more closely on one side than on the other. Sometimes, also, the palate of the paralyzed side hangs somewhat lower than normal, and moves less freely during respiration; under such circumstances the uvula hangs somewhat obliquely, with its point directed sometimes towards the paralyzed, sometimes towards the unaffected side of the body.

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[1] The various deviations from the normal type will be treated of in detail in connection with the subject of the localization of lesions.

[2] *O. Berger*, Die Lähmung des Nervus Thorac. longus. Breslau, 1873.

Such are the usual characteristics of hemiplegia. All the other motor nerves of the body are much less often paralyzed than those mentioned; yet there is not a single one that may not, as the seat of the lesion varies, be affected. At the head of the list, in point of frequency, comes the hypoglossus; it has already been mentioned that in most cases of apoplexy a certain degree of difficulty in the movement of the tongue is present at the outset, but that, as a rule, it quickly disappears; yet cases are met with where it remains permanently. The nerves supplying the muscles of the eye are affected almost as often as the hypoglossus; this results, as a rule, in strabismus, with its attendant disturbances of vision, which persist only for a few days. Still more common are changes affecting the size of the pupil on one side, which also disappear after a short time. Sometimes a paresis of the upper eyelid remains permanently. A special form of the affection in which the paralysis of the oculo-motor is on the opposite side to that of the limbs, will be spoken of later. Unilateral paralyses of the vocal cords, or of the pharynx, are exceedingly rare; functional impairment of the spinal accessory nerve is still more so.[1]

Certain exceptions have been observed to the rule according to which hemiplegia affects the side of the body opposite to that of the lesion (not to speak of certain special forms of paralysis, to be discussed below). The number of these exceptional cases is infinitely small as compared with those which observe the general rule, but it is nevertheless unquestionably true, and not merely a matter of inaccurate observation, as Romberg believed, that now and then the only discoverable lesion in the brain is situated on the same side with the paralysis of the extremities.[2] It is very difficult to give a satisfactory explanation of this occurrence, standing in opposition as it does to the settled teachings of anatomy and physiology; one theory, proposed some time ago, scarcely need be mentioned at present, as it is utterly untenable. That of Morgagni and Brown-Séquard, that in such cases the normal decussation of the pyramids is wanting, or that of Schiff, that a redecussation afterwards takes place, are simply hypotheses, *ad hoc*, without proof. Ambrosi believes that the circumstances under which this hemiplegia on the same side occurs are such that it is due, not to the primary lesion itself, but to secondary changes, especially of the nature of œdema and softening, so extensive as to involve the ganglia on the other side of the brain. This was in fact found to be the case in one instance ; but the corpus striatum on the same side, *i. e.*, on the side of the lesion itself, was also soft and œdematous ; and the question therefore remains, why this was not also the starting-point of paralytic symptoms affecting the opposite side of the body ?

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[1] A detailed account of the symptoms arising from the paralysis of special nerves will be treated of in another part of this work.

[2] Collections of cases of this kind have been made by Morgagni, Burdach, Dechambre, Andral, Nasse, Ambrosi. The latter has very recently reported such a case, which was observed in Leyden's Clinic (Ueber gleichseitige Hemiplegie. Inaug. Dis. Königsberg, 1867).

Besides occurring in this typical hemiplegic form, the paralyses from cerebral hemorrhage may sometimes appear in other shapes. These differences are dependent only upon the varying seat of the extravasation. Thus sometimes bilateral paralysis, paraplegia, is observed ; this is due either (in very exceptional cases) to hemorrhages occurring simultaneously in both hemispheres, especially in the central ganglia, or to extensive lesions in the pons or medulla oblongata. Under these circumstances it could scarcely happen that life should be long preserved. The statement, that a lesion of one side of the brain above the pedunculus cerebri has been known to cause a real bilateral paralysis, is so utterly in contradiction to well-recognized anatomical and physiological facts, that it is much easier to believe that some other lesion existed, but was overlooked at the autopsy (the absence of secondary changes being taken for granted, such as œdema, and the like, extending to the other side of the brain).

Cases of so-called *alternate paralyses* (first named and studied by Gubler), in which a unilateral, intra-cerebral hemorrhage, not in a position to compress the nerve trunks at the base of the skull, causes paralysis at once of certain muscles on the opposite side of the body, of others on the same side, deserve special notice. When the extravasation is in the crus cerebri (vide below), the muscles of the extremities and the face of the opposite side are affected, and those supplied by the motor-oculi on the same side ; in this case the motor-oculi, lying as it does in immediate contact with the crus, is injured, not at its centre, but in its course. Further, lesions of the pons may cause another species of alternate paralysis, affecting the extremities on the opposite side and the face on the same side. The special features of the facial paralysis in this case and the probable cause of this so-called alternate arrangement will be discussed more in detail below. Finally, a third form of alternate paralysis is sometimes, though very rarely, seen, in which, in consequence of a single lesion, the upper and lower extremities of opposite sides are affected; in this case the seat of the lesion is in the upper part of the medulla oblongata, at the point where the decussation of the nerve fibres for the lower extremities has already occurred, but just above the point of decussation for those of the upper extremities. (Vide Schiff's Physiologie des Nervensystems.) So far as we know, no uncomplicated case of hemorrhage, resulting in this form of paralysis, has been observed.

It occasionally happens, also, that the distribution of the paralysis is very limited, *confining itself to individual nerves;* these are, under such circumstances, always cranial nerves. We are not aware that cases of paralysis, restricted to single nerves of the extremities—for example, the median or musculo-spiral—have ever been reported as a consequence of hemorrhage. It happens indeed comparatively rarely that even one extremity alone is attacked without the other being at all affected. Paralysis of individual cranial nerves is, nevertheless, met with not only as a prodromal symptom of an apoplectic attack, to be followed, later, by more wide-spread symptoms, but also, from time to time, as a permanent symptom by itself. In most of the cases of this kind, hitherto observed, the facial nerves have been the ones affected. Paralysis, thus limited to the facial nerve, has been seen in connection with extravasation in the optic thalamus, and in the corpus striatum (for example, by Duplay[1]); Cruveilhier reports this as having happened to Dupuytren, in consequence of a hemorrhage—no paralysis of any other part following later; Chvostek[2] describes a case in which disturbances of speech and a feebleness of the extremities on the left side, present at first, disappeared almost entirely after a few days; on the other hand, the left facial nerve remained almost completely paralyzed (even the branch to the orbicularis palpebrarum), and the autopsy disclosed the presence of a hemorrhagic cyst in the right nucleus lenticularis.

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[1] Union Méd. 1854.

[2] Oesterr. Zeitsch. f. pract. Heilkunde. 1870. No. 35.

Although the voluntary control of the limbs is entirely lost, yet there are various conditions under which involuntary movements may take place in them. Thus well-marked, associated motions are not unfrequently seen, having their origin especially in strong mental excitations and in certain involuntary reflex processes. Thus the muscles of one-half the face, usually completely relaxed, may, in connection with those of the unaffected side, perform motions necessary to changes of expression, sometimes contracting even more strongly than do the healthy muscles, as in laughing, and the like; in other cases, on the contrary, the difference between the two sides of the face comes out all the more strongly under changes of expression.[1] Under the influence of emotional excitement, such as anger, the paralyzed extremity may be more forcibly moved than the other; when the patient coughs, gapes, or sneezes, during micturition or defecation, the paralytic member may be jerked strongly into the air, or may fall into a state of convulsive twitching; if a certain amount of contraction is already present, it becomes still more decidedly marked; and yet, withal, the utmost effort of the will may be unable to provoke the least motion.

Attention may here be drawn to a fact, which was known also to the older writers, namely, that when hemiplegic patients are brought under the influence of strychnia, the muscular contractions occur in the paralyzed parts long before any symptoms of the poison show themselves in the unaffected extremities.

Another form under which these associated movements are seen, is that in which a movement, performed voluntarily by the unaffected extremities, is imitated by the corresponding muscles on the paralyzed side. This form is less common than the first mentioned, and is most clearly marked in cases where permanent contractions have already made their appearance. Westphal reports them as being especially noticeable in persons who have been hemiplegic from youth. Still less common is a third form, which occurs occasionally in cases of apoplexy, as has been pointed out particularly by Hitzig.[1] When, namely, during the period in which the power of voluntary motion is returning, the patient wishes to make any particular movement—for example, flexion of the forearm—he seems compelled to exert all his strength in the effort, as if obliged to overcome some powerful resistance. Close examination shows that this analogy is in fact a just one, since in such cases not only the biceps, but the triceps as well is found strongly contracted. Not only the contraction of the muscles immediately necessary for the desired movements, but also that of their antagonists, is thus seen to be excited by the voluntary effort in a degree out of all proportion to the end to be accomplished. We can confirm Hitzig's statements on this point.[2]

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[1] This associated action of the paralyzed half of the face in expression, in cases where the voluntary control over it is lost, is seen in connection with lesions in certain definite places, as will be pointed out more at length in another place.

We would refer, in this connection, to still a fourth form under which the associated movements may show themselves in cases of hemiparesis. When an apoplectic patient, who has ceased to be completely paralyzed, innervates the muscles of the unaffected side—if he extends the fingers, for example—the paretic muscles remain at rest; if, on the other hand, he extends the partially paralyzed fingers, which is accomplished only slowly and with effort, those of the unaffected side perform the same motion. Except during the time immediately after the attack, probably very few cases of hemiplegia from cerebral hemorrhage occur in which one variety or another of these associated movements is not met with to a greater or less extent.

It is much less common to meet with *reflex movements*, properly speaking. When they do occur, it is in obedience to the general laws governing such movements, *i.e.*, they follow sensitive excitations (especially percussion, Uspensky), which are unfelt by the patient; and they do not confine themselves to the paralyzed parts, but may involve the other limbs as well.

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[1] Arch. f. Psych. u. Nervenkrankh. III. Bd.

[2] The same condition is met with in the course of other paralyses—for example, those following typhoid; vide my article, "Ueber centrale Irradiation des Willensimpulses." Griesinger's Archiv für Psychiatrie und Nervenkrankheiten. Bd. III.

We are as little able to point to thorough, systematic investigations serving to explain this fact, to-day, as Hasse was, twenty years ago.

The following are the different forms under which it is possible for these movements to make their appearance: sometimes the feeblest excitations call out energetic contractions in completely immovable limbs, while at other times the strongest excitations are without effect; again, it sometimes happens that excitation of distant parts calls out these reflex motions. (Marshall Hall reports a case, observed by Holland, where the dressing of a seton-wound, alongside of the lumbar vertebræ, provoked a powerful jerk of the right (paretic) arm.)

In still another set of cases moderate pinching of, for instance, the right, totally paralyzed arm excites contractions in the left, unaffected, arm; somewhat severer pinching, similar movements also in the left leg; and still stronger pinching, the same symptoms in the right, paretic (*i. e.*, not entirely paralyzed) leg; while all this time the right arm itself remains at rest, irritate where and how one will.

In our experience these results remain the same, whether the sensibility of the skin is increased, diminished, or normal, or whether the paralysis of motion is attended or not by pain. As a rule, however, it happens that the abnormal increase of reflex irritability is met with, especially in the period which begins three or four weeks after the attack, and lasts for three or four months, although it is sometimes seen as late as after several years. As already indicated, it is impossible to say, at present, what it is that determines the variations in reflex irritability, mentioned above: whether the position of the lesion is of significance, or the influences, of which we shall speak in discussing the spasmodic contraction of muscles that is sometimes observed, or the removal of the "inhibitory" influence of the brain (Setschenow's inhibitory-centres), as Uspensky suggests, or, finally, whether the secondary degeneration of the damaged nerve-tracts is the cause, as Hasse believes, of the diminution of irritability where that exists. The central seat of these reflex processes is, in all probability, to be sought in the medulla oblongata.

As in connection with various other local diseases of the brain, so also in connection with hemorrhages, the tonic muscular contractions which often occur (the so-called *contractures*) are of great importance. These contractures may be divided into three classes, according to the periods in the course of the affection at which they occur: 1, those which immediately accompany the hemorrhage; 2, those which appear early, a few days after the attack; and, 3, those which take place later during the paralytic stage.[1]

We have already spoken of the first class in describing the occurrence of the hemorrhage; the contractures of this order disappear, almost without exception, after a few days. Those of the second class have also been referred to in connection with the symptoms of the reactive inflammation of the tissues about the blood-clot. They are associated, in point of time, with this latter process, *i. e.*, they occur in the paralyzed parts a few days after the occurrence of the hemorrhage, and disappear again after a short time. The theory, proposed by Todd, in regard to the origin of this class of contractions, is the one usually accepted, viz., that the inflammatory process exerts an irritant action upon the cerebral nerve tracts. It must, however, be acknowledged that the manner in which this assumed irritation is produced is as yet by no means clear.

Far the commonest form of contracture is that which occurs in the later stages of the affection, affecting the paralyzed limbs; it is rarely wanting in cases where the life of the patient is long preserved, and his condition remains stationary, although, to be sure, it is often so trifling, causing sometimes only so light a flexion of the fingers as to escape notice in a superficial examination. Nevertheless cases certainly are met with, especially where the symptoms are improving, in which no trace of this condition is to be seen. It probably never happens that all the paralyzed or paretic muscles are affected with equal severity in this way. As a rule, the upper extremity is regularly attacked, the lower less often; a contracture confined to the lower extremity has never, so far as we know, been observed. It has also not yet been observed with certainty in the respiratory muscles, nor in those of the trunk; it is met with sometimes in the muscles of the face, and I have seen a case in which the sterno-mastoid, the scaleni, and the trapezius were attacked, causing a permanently oblique position of the head. The resulting position of the limbs under these circumstances is not always the same; the most common is flexion at the elbow, very rarely extension; next in order of frequency occurs flexion and pronation of the hand and fingers (observed by Bouchard in twenty-two cases out of thirty-one); sometimes, however, flexion with supination and sometimes extension of the hand and fingers is met with. The position of the shoulder varies; the arm is often adducted, rarely abducted; the foot is sometimes extended, sometimes flexed, the knee commonly flexed. The statistics given by Bouchard are not sufficiently full to be used in drawing general conclusions. The intensity of the contracture varies; it is sometimes so slight as to be overcome without difficulty; in other cases it resists the strongest traction without yielding, though the stretching may be very painful. The intensity and character of the contraction are not always proportional to the permanency of the paralysis; sometimes no contraction is present.

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[1] We shall refer further on to the apparent contractures caused by diseases in the joints.

Hitzig has lately called attention to a peculiarity in the behavior of these hemiplegic contractures, which, to be sure, had been remarked upon by isolated observers, but had never received its due share of attention—and that is, that they become absolutely fixed and immovable only after having existed for several years. In the early period of their existence, the contracted muscles may relax, so as to allow the paralyzed limb to reassume its normal position. This occurs when the patients have remained quiet and at rest for some time, especially if they have been lying down, without moving. Since these conditions exist pre-eminently during sleep, it sometimes happens that in the morning, on awaking, such patients find the muscles, which were strongly contracted the evening before, relaxed, and in part under the control of the will; so soon as they make any active attempt to move, however, the stiffness returns at once. I am able to confirm this observation of Hitzig's; in fact, since my attention was first attracted to the matter, I have observed the condition to which he refers in every case that I have examined. It has already been remarked that the contractions already existing become still firmer under the influence of associated movements. The period at which this muscular rigidity appears may vary between wide limits: we have never observed it before the second month, sometimes not until the fifth month.

In attempting to answer the question, what is the cause of these contractures, we may pass over the explanations offered by the older writers, and need discuss here only two, proposed in recent times. Bouchard believes that they have to do with the secondary connective-tissue changes in the spinal cord, which he supposes to act as an irritant to the nerve-fibres. Hitzig criticises this theory, by pointing to the fact that the contractures often vary in intensity, that they may finally disappear altogether, and that the muscles of the extremities differ among themselves and from those of the trunk, in their liability to be affected. He is inclined to look upon them as the expression of repeated, abnormal associated movements. Hitzig's view, so far as it admits of brief definition, is as follows: even under physiological conditions, the effort to contract certain definite muscles excites associated contraction in others as well. The more intense this effort, the more widely it diffuses its action through the higher cerebral centres which stand in immediate relation to the will. According to the teachings of physiology, there exist, in the lower (more peripheral) sections of the encephalon, ganglionic centres within which the peripheral nerves which terminate in them are grouped together for physiological ends, in accordance with a definite plan.

It is, then, conceivable that strong efforts of the will, having their anatomical starting-point in the cortex cerebri of the uninjured side of the brain—if, by virtue of the irradiation mentioned above, they reach these centres, which we may imagine to have been thrown into a state of abnormal irritability through the agency of the hemorrhagic attack—should, instead of confining their action to the particular nerve-tracts, for which they were destined, excite others in the neighborhood as well, and thereby call out involuntary associated movements in the paralyzed muscles; and, further, that in consequence of the supposed irritability of the centres, these associated movements should be at times exceedingly strong, and should finally pass over into the condition of contracture already described. The nature of this state of irritability is unknown.

This theory of Hitzig's seems to us quite reasonable, and worthy of further investigation.

The so-called *hemiplegia spastica infantum* (Benedict) is characterized essentially by contractures of the kind described, occurring in hemiplegic children.

According to Benedict, a peculiar feature of these cases is, that the patients are able to extend the paretic half-flexed fingers if the flexion is made still greater by the hand of another person; and that, in like manner, when the patients wish to extend their own fingers, they instinctively make a movement of flexion, in connection with which the desired extension takes place.

Besides the symptoms already described, which occur with almost unvarying regularity, these cases present evidences of other motor disturbances, which a closer examination would certainly discover to be more common than is supposed. They do not make their appearance at that period in the disease when the paralysis is complete, but only when the power of voluntary motion has been restored to a certain degree. The movements of the patient at this stage, though usually slow and weak, may be otherwise perfectly normal. In certain cases, however, the motions of these paretic extremities may be of an *ataxic* or *choreiform* character. We have seen one case where the symptoms (the diagnosis was not tested by post-mortem examination) pointed distinctly to hemorrhage into the corpus striatum, in which well-marked, unilateral ataxia was present, of the same character as that occurring in tabes dorsalis.

Charcot[1] has recently reported several cases of unilateral tremor and choreiform movements, where, at the autopsy, changes were found in the thal. opt., corp. striatum, and neighboring parts of the opposite side of the brain, and Leyden[2] observed a case of pronounced, typical ataxia, without paralysis, following the occurrence of softening at definite points, within the pons Varolii.

We must leave it to future investigations to determine whether or not it is peculiarities with regard to position that make one hemorrhage more likely than another to cause disturbances of motion of this kind.

Leyden[3] reports the case of a patient in whom, in consequence of a hemorrhage (or softening, believed to be situated either in the pons, cerebellum, or corp. quad.), the above-mentioned ataxia was present in all four extremities, and in whom, moreover, voluntary movements were performed with decidedly less rapidity than normal; there was evidently no retardation in the conduction of sensitive impressions, and the symptoms appeared to be due to a *retardation in the conduction of*

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[1] Gaz. Méd. 1873. No. 36.

[2] Klinik d. Rückenmarkskrankheiten. I. Th. S. 116.

[3] Virchow's Archiv. 46. Bd

motor impulses, through the central nervous system. **It is as difficult to explain this phenomenon as to explain the ataxia.**

The law governing the electrical reaction of the paralyzed parts is to be regarded, at any rate from a clinical point of view, as almost exhaustively understood. Of special importance, in this connection, is the fact that in paralysis due to cerebral hemorrhage, in common with the intra-cerebral paralysis in general, the reaction of the muscles under both the galvanic and the faradic current remains unchanged. The truth of this important law has been proved by repeated observations, and is daily to be confirmed anew, so that it is only worth mentioning as a matter of historical interest that Duchenne was the first to formulate it in this shape—Marshall Hall having previously stated that in the cerebral paralyses the parts showed an abnormally great irritability to electricity. Although the paralysis may have existed for years, contractions of the affected muscles follow the application of either kind of electricity, just as in health. This general rule has, however, certain limitations. It sometimes happens that the irritability is diminished, occasionally to a marked degree. In making this latter statement, we exclude from consideration the cases of intra-cranial, not intra-cerebral, paralysis, such as have been carefully described by Ziemssen,[1] in which the cranial nerves, especially the facial, are injured in their course, after leaving the brain, by some morbid process at the base of the skull. In these cases the electrical reaction is the same as in cases of traumatic affections of peripheral nerve-trunks in general, *i. e.*, it diminishes progressively, even to complete disappearance, and that too in a relatively short time. (For the details of this process, vide the appropriate section of this work.) Further, Benedict,[2] and still more Rosenthal, followed by Duchek and others, have called attention to the fact, that even in a case of intra-cerebral paralysis a notable diminution in the irritability of the facial nerve may at times occur, especially when the lesion is in the pons Varolii. Benedict states, moreover, that when the seat of the lesion is in the pedunculus cerebri, this same diminution of irritability occurs after the lapse of some time. It is also possible that, as Eulenburg believes, a similar diminution of irritability may follow secondary degeneration of the spinal cord, though the proof of this has yet to be given. Finally, the same result may be brought about by an atrophic process in the muscles themselves, which of course must impair the strength of their contraction.

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[1] Virchow's Archiv. 13. Bd.

[2] Elektrotherapie.

Rosenthal affirms that the electro-muscular sensibility is often markedly diminished, although the motor irritability may be entirely intact, and that this is especially true when the skin is at the same time anæsthetic.

Increase of electrical irritability is met with much less often than the slight diminution described. Apart from the apparent increase due to diminished resistance of the tissues on the paralyzed side (for example, from absorption of the subcutaneous fat) which is occasionally seen, this phenomenon is most frequently observed, though even then only to a moderate degree, in fresh cases of paralysis characterized by symptoms of motor irritation. Crossed reflex contractions sometimes occur, according to Benedict, in consequence of hemorrhage into the medulla oblongata in such a position as to injure the nucleus of the facial nerve. Under these circumstances, electrical irritation of either side of the body, the paralyzed or the unaffected, may excite reflex contractions in the muscles of the opposite side of the face.

The paralyses, which in their essential features we have just described, form the most constant and permanent symptoms of hemorrhage. In fact, the cases in which no paralytic symptoms whatever are present are exceedingly rare. The constancy of their occurrence is explained by the fact that the motor nerve-tracts happen to pass directly through the regions which are the favorite seat of hemorrhages (vide above), and it serves as an additional argument in favor of the view, to which experiments also have lent support, that lesions in many parts of the brain, even within the hemispheres, may cause motor-paralysis, or paresis.[1] I regard it as unquestionable that the entire absence of paralysis indicates that the hemorrhage is situated at some point which is removed from motor tracts; as to the exact position of these points, that is a matter for future investigations to discover.

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[1] Virchow's Archiv. 57. 58. 60. Bd.

Disturbances of Sensibility.

It has long been known, and is universally recognized, that in cases of hemorrhage, as in all the localized affections of the brain, changes of sensibility are vastly less common than those of the motor functions. In many cases, the anæsthesia is, from the first, very slight, or if well marked in the beginning it may soon disappear again, or it may have been entirely absent; it happens only exceptionally that the paralysis of motion and that of sensation keep pace with one another, or that the latter is the more prominent symptom of the two. We will now consider these points more in detail. As a general rule it happens that, immediately after the attack, the sensibility as well as the motion of the paralyzed half of the body is seriously impaired; but just as the motor paralysis is much more extensive in the very beginning than after the lapse of a few days, so also the anæsthesia for the most part soon disappears, the reasons for the latter occurrence being probably the same as those suggested above for the former. In other cases, the disappearance of the anæsthesia takes place more slowly. The recovery occurs centrifugally, *i. e.*, the sensibility of the hand and foot remains longer impaired than that of the arm and thigh.

The return of cutaneous sensibility to its normal state, whatever may have been its condition at the outset, may or may not be absolutely complete. From my own experience, I am inclined to think that an incomplete restoration is oftener met with than is generally believed. It is true that the patient may feel the lightest prick with the needle distinctly on both sides; but if the character of the two sensations be carefully compared, they will often be found to be duller on the paralyzed side, or the difference between the touch with the head and that with the point will be found to be less easily recognized. Lesions of the nucleus lenticularis and corpus striatum (as will be shown below), which are the commonest of all, are not accompanied by disturbances of sensibility. The reason that, in spite of this fact, it is so common to find slight disturbances of this kind, is that the extravasations are rarely limited to the above-mentioned ganglia, but overstep their borders in one direction or another.

When the sensibility is diminished at all, it happens by far the most frequently that it is equally affected in all its departments. Occasionally, however, it happens that cerebral hemorrhage, like other cerebral diseases, gives rise to a paralysis of certain kinds of sensation only (Puchelt,[1] Landry,[2] Mosler,[3] Berger,[4] and others).

It is not as yet determined exactly where the extravasation must be situated in order to be followed by this result; probably, however, in the hemispheres or the cortex.

With regard to the *distribution* of the anæsthesia, it may be confined to limited regions—for example, the face—while in other cases it is precisely the distribution of the trigeminal nerve which remains unaffected; as a rule, however, its distribution is the same as that of the paralysis, taking in one entire half of the body, including the trunk; even the tongue often shows a diminution of sensibility upon one side. According to E. H. Weber, areas of disturbed and of preserved sensibility may be found side by side, a fact which may be explained on the supposition that certain parts of the central nerve tracts remain uninjured.

It has already been pointed out that now and then a high degree of anæsthesia remains *as a permanent symptom.* Türck[5] was the first to point out the fact, afterwards substantiated by Rosenthal, that in such cases the extravasation often occurs within definite limits, viz., in a region which includes the posterior part of the inner capsule, the innermost division of the nucleus lenticularis, the external superficial layers of the thalamus opticus, and the adjacent portion of the corona radiata. Since, in the cases in question, more than one of these parts have invariably been involved, it is not possible to decide which of them is the one chiefly concerned.[6]

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[1] Ref. in Canstatt's Jahresbericht. 1845.

[2] Arch. Génér. de Med. T. XXIX. and XXX.

[3] Berl. klin. Wochenschr. 1868. No. 39.

[4] Wiener med. Wochenschr. 1872.

[5] Sitzungsbericht der Wiener Akademie. 1859. Bd. XXXVI.

[6] Recent experiments by Veyssière (Arch. de Physiol. Normale et Pathol. 1874, p. 288) confirm the clinical observations, *i.e.*, that destruction of the tissues in this neighborhood causes anæsthesia. But even these experiments do not enable us to say precisely what nerve tracts are the ones chiefly concerned.

Besides this, marked and lasting anæsthesia may follow localized lesions in the pedunculus and pons Varolii (vide below).

The disproportion between motor and sensitive disturbances has long been a matter of common observation. An analogous disproportion is found also, as is well known, in cases where the spinal cord or peripheral nerve trunks are subjected to pressure. But how is this to be explained? Evidently only through the assumption that in those parts of the brain which are the favorite seats of the extravasation, few if any conductors of sensibility are present; for we know that when actual destruction of such conductors has taken place, as, for example, in lesions in the pedunculus cerebri, unmistakable anæsthesia is the result; the experimental observation also, that artificial destruction involving the nucleus lenticularis, thalamus opticus, or nucleus caudatus are followed invariably by disturbances of motor but never of sensitive character, is in harmony with this view. We have already indicated above our belief as to the origin of the alterations of sensibility which immediately follow the attack.

The list of sensitive disturbances is not yet exhausted. Besides the anæsthesia, we may meet with hyperæsthesia, or, more properly speaking, *hyperalgesia;* but in the cases of which we speak there is no real exaltation of the sensitive functions, but a condition is present in which even a light touch is felt as pain. M. Rosenthal describes cases in which anæsthesia, which was present for a few weeks after the attack and then disappeared, was not immediately followed by a condition of normal sensibility, but, for a time, by a hyperalgesia of this kind. I have observed other cases in which, after several, for example, eight weeks succeeding the attack, the skin, which had already been moderately anæsthetic, became excessively sensitive to pressure and pricking with the needle, and remained so for weeks or months, finally returning to its previous anæsthetic condition. In still other cases I have seen this hyperalgesia exist side by side with moderate anæsthesia, even into the third or fourth year. The patients not unfrequently declare that the severe pain—for example, from pinching—does not remain limited to the part irritated, but extends over the entire extremity or the entire half of the body on the same side. In such cases the above-mentioned increased irritability is sometimes present, though often wanting.

Besides the hyperalgesia from external irritation, *spontaneous attacks of pain* sometimes occur. We do not refer here to the pain mentioned above as coming on some days or weeks after the attack, and due to the reactive inflammation in the neighborhood of the lesion; but to those cases in which the totally or partially paralyzed limbs are for years the seat of neuralgic although diffused pains, which may last for a time without change, or may be marked by periodical exacerbations (especially in damp weather). In other cases, as Brown-Séquard, Charcot, and Hitzig have pointed out, the pains are often felt principally in the joints, especially the shoulder-joint.

Finally, the abnormal sensations, which are observed from time to time, deserve mention, such as those of stiffness, formication, and prickling ("going to sleep").

It is not, at the present time, possible to give a satisfactory explanation of the pathogenesis of the hyperalgesia and of the persistent neuralgic pains. It is hardly fair to attempt to account for the latter symptom by assuming the existence of an inflammatory process, continuing for years, through which the extremities of the severed sensitive nerves are irritated. At the most, it might be regarded as possible that an influence of this kind was exerted by the products of such an inflammation, which would make these cases analogous to those in which neuralgia follows upon the section of peripheral nerves. This supposition would accord with the fact that, at least in cases which I have observed, a greater or less degree of cutaneous anæsthesia is likewise present. I would also call attention to the fact that *the severest instances of these diffused pains which I have seen have occurred in connection with œdematous swelling of the skin*, to be described later. Is it possible that a causal connection exists between the two conditions? The pains limited to the joints are associated with inflammatory processes in them, which will be discussed further on.

Trophic and Vaso-motor Disturbances.

It sometimes happens that immediately after an attack of hemiplegia a state of things exists in the paralyzed limbs which is doubtless to be referred to an affection of the *vaso-motor nerves;* these extremities, namely, in spite of the paralysis, are redder and warmer than their fellows. According to my observations, which accord with those of others, the difference of temperature amounts usually only to from a few tenths to a little more than one degree; sometimes, however, to several degrees. As stated, this difference in temperature and color between the paralyzed and non-paralyzed extremities is not always present.

Side by side with these changes, another symptom is often, but not always, present, which was recently described by some French observers as if newly discovered, although in fact it had been recognized before, and which, to judge from my experience, is so common that I consider Hitzig as justified in saying that it occurs as a rule in cases of complete hemiplegia. This is a condition of swelling and general enlargement of the extremities, which an attempt to pinch up the skin readily shows to be due to an affection of the cutaneous tissues, apparently of the nature of *œdema.* This may come on after twenty-four hours, and may persist for a long time. The heat and redness which accompany this œdema generally diminish and disappear after some weeks or months, and with them the œdema itself. In contradistinction to the condition which obtains at the outset, the temperature of the paralyzed limbs at a later period may be no greater than that of the unaffected parts; sometimes, indeed, several degrees less, so as to feel icy cold to the touch. Yet, in spite of feeling colder, the skin may present a purplish, cyanotic appearance (it has been mentioned above that this condition is often accompanied by pain). In the same category with these phenomena belong the changes in the *perspiratory function of the skin.* In the early stages, so long as elevation of temperature and œdema are present, patients usually suffer at the same time from profuse sweating of the affected side. This tendency disappears after a time; but so long as they remain œdematous, the paralyzed extremities may be more moist than those of the other side, even though they are very cold. Sometimes the reverse condition is present, the skin tending to be dry and scaly.

Still other phenomena, to which attention has recently been called anew, may be mentioned in this connection. We have referred above to the experimental observations of Schiff and of Brown-Séquard, that lesions in certain parts of the brain may be followed by hyperæmia or hemorrhage in the pleura and lungs of the paralyzed side. We sometimes find the same condition at post-mortem examinations of persons dying from cerebral hemorrhage, and at times, associated with it, hemorrhages in the corresponding kidney and in the skin, as well as marked injection or ecchymosis of the pia mater on the side of the hemiplegia—*i. e.*, the side corresponding to the unaffected half of the brain (Charcot, Ollivier, Bennet, and Barety[1]). Finally, *alterations of the pulse* are to be mentioned. It was known to the older observers that a difference sometimes existed between the radial pulses of the affected and non-affected sides, as indicated by the term pulsus differens. Wolff and Eulenburg have discovered by sphygmographic investigations that the amplitude of the pulse on the paralyzed side is diminished, and that, furthermore, other peculiarities in the lines of ascent and descent are met with that are due to a diminished contractile power of the walls of the vessels.

All the phenomena described are plainly due to injury inflicted upon vaso-motor nerve tracts. We need not go into this matter in detail, as the physiological facts on which the statement rests are well known. In connection with œdema of the skin alone, we would call attention, among others, to the experiments of Ranvier[2] (confirmed by Emminghaus), who found that ligation of the vena cava inf. in dogs would cause great diminution of temperature, but no œdema, in the hind-legs; whereas, if after this operation the sciatic nerve, which contains the vaso-motor nerves of the leg, were cut on one side, the paralyzed limb became very hot, vascular, and after a few hours the seat of marked œdema.

At the same time the problem still remains unsolved with regard to this whole series of phenomena, how it is possible that lesions in so many different parts of the brain should give rise to vaso-motor disturbances. We are, in fact, still utterly ignorant as to the exact course of the intra-cerebral vaso-motor and trophic nerve tracts.

It happens occasionally that hemorrhages, like other localized diseases of the brain, give rise to acute *bed-sores*, as has been pointed out especially by Charcot. These sores are met with only on the paralyzed side, generally in the middle of one of the nates, less often at the knee or on the heel. As a rule, they begin with an erythematous reddening of the part, showing itself from two to four days after the apoplectic attack; on the next day bullæ are apt to appear, and sloughing of the skin follows quickly. Charcot regards the development of a bed-sore under these circumstances as a highly unfavorable symptom, believing that it indicates almost invariably the approach of death.

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[1] It may be here remarked that Ollivier observed the occurrence of a moderate degree of albuminuria immediately following the attack in several cases of severe hemorrhage of varying seat; in one case also diabetes. In only two of these cases was the hemorrhage found in the pons Varolii or medulla oblongata. The autopsies revealed renal congestion, together with sub-capsular hemorrhage and renal apoplexy. (Archiv. Génér. 1874, Fevrier.)

[2] Comptes Rendus. 1869.

In Charcot's opinion, which we regard as the correct one, though we have not the space to adduce here the evidence upon which it is founded, this bed-sore does not result from vasomotor changes, but from some affection of some system of nerves having trophic functions.

Further trophic changes present themselves under the form of disturbances in the growth of the *nails* and *hair* on the paralyzed extremities. The former become yellowish, ridgy and brittle, and abnormally curved in both directions; the hair grows thicker and longer. Hypertrophic changes occur also in the skin, which are especially worthy of notice. They involve both the cutis and the panniculus adiposus, so that a fold pinched up by the fingers is thicker than normal.

In many cases of hemiplegia *inflammations in the joints* also occur, limited to the paralyzed side. They are directly or indirectly connected with the cerebral lesion, and may follow extravasations of blood, though they are oftener due to softening. This condition is met with both in an acute and in a chronic form. The former (Scott Alison, Brown-Séquard, and especially Charcot) begins from a few days to four weeks after the hemorrhage, sometimes still later; the joint becomes reddened, hot, and swollen, and after death acute synovitis, often with considerable exudation, is discovered. It is almost exclusively the larger joints which are thus attacked.

On the other hand, Hitzig has described a chronic form of this articular inflammation, which is scarcely ever met with anywhere but in the shoulder-joint. The joint becomes sensitive to pressure and to passive motion, and at the same time almost immovable; crepitation may be felt and heard within it, and it takes a position of semi-dislocation, characterized by flattening of the shoulder and dropping of the head of the humerus. This description is confirmed by my own experience.

Charcot regards the first mentioned form of inflammation as being of neuroparalytic or, rather, neuro-trophic origin, dependent upon some affection of the trophic nerves. Hitzig, on the other hand, considers the primary factor in the production of the inflammation in his cases to be a sub-luxation of the humerus consequent upon the paralysis, and regards the relaxation of the muscular walls of the blood-vessels, together with the accompanying diminished power of resistance on the part of the tissues as having only an accessory influence. We cannot discuss here the pros and cons of this question; but we recognize the fact that the articular inflammation may be of two distinct kinds, so that both of these opinions may be correct. (It is, for example, impossible to explain on Hitzig's theory cases such as one reported by Alison, where, a few days after the paralysis, the joints of the knee and foot became inflamed.)

It is a very striking fact, though one of frequent occurrence, that the paralyzed limbs of hemiplegic patients, in spite of having been absolutely immovable, sometimes for years, do not become wasted (sometimes, indeed, in consequence of the hypertrophy of the skin they appear larger than those of the unaffected side), in which respect these cases differ essentially from many forms of paralysis of peripheral or spinal origin. In accordance with the theories of the day, this is usually explained on the assumption that the "trophic" fibres escape uninjured. If this position is justly taken, it must certainly be regarded as doubtful whether the explanation given for the formation of bed-sores and the disturbances in the growth of the hair and nails is a correct one, and the same might be said of Charcot's theory as to the origin of the inflammation in the joints. When hemorrhages occur in persons whose growth is not yet complete, especially in early childhood, then certainly a marked degree of atrophy takes place and the growth of the paralyzed limbs remains defective; they become shorter and smaller than their fellows, from imperfect development of all their tissues, even the bones. In spite of this, it is striking to find the electrical irritability of the muscles preserved, sometimes even apparently increased, owing to the fact that the subcutaneous layer of fat is thinner than normal.

Disturbances of the Special Senses.

Symptoms of this kind, like the disturbances in the sensibility of the skin, occupy a much less prominent place than the motor paralyses. No thoroughly reliable cases are reported of *anosmia* (paralysis of the olfactory nerve) occurring as a consequence of cerebral hemorrhage; a diminution of the power of smell on one side is more often met with as a consequence of the partial closure of one nostril from the paralysis of the facial muscles. It is not uncommon to find, during a short time after the attack, a diminution of the faculty of *taste* limited to the forepart of the tongue on one side; this is due to an affection of the chorda tympani. This condition occasionally persists for a long time, but usually disappears very soon. Paralysis of the glosso-pharyngeal nerve of one side, causing permanent loss of taste over the posterior half of the tongue on one side, has never been observed with certainty, as far as we know, as a result of an attack of hemorrhage. The *hearing* sometimes remains slightly affected, but complete unilateral deafness is rarely met with.

We can say but little that is definite with regard to the permanent disturbances of *vision*. It is certain that they are not of common occurrence, although we have no statistical proof of the statement to offer. It is still somewhat doubtful whether optic neuritis ever follows simple cerebral hemorrhage; it certainly happens but seldom. Hemiopia, on the other hand, has been shown, by recent investigations, to be of frequent occurrence.[1]

In four cases reported by Cohn, without autopsy it is true, but where the symptoms seemed to justify the diagnosis of hemorrhage (or, in one case, perhaps embolism), a bilateral, left hemiopia was present, associated in two of the cases with paralysis of the left side of the body; while in one case there was no motor disturbance, and in one paralysis of the right side came on after the appearance of the hemiopia.

Disturbance of the Mental Functions.

It happens only in a small fraction of the cases that persons, afflicted with cerebral hemorrhage, regain totally the use of all their mental functions. By far the greater majority of them remain for the rest of their life more or less deficient in this respect. Memory is the faculty most prone to fail, especially as regards the retention of recent impressions ; the patients forget the affairs of the present moment or that which has just passed, while they remain able to recall every experience and impression of their former life, even those of childhood, with unimpaired distinctness. The reflective faculty or power of judgment suffers less often ; yet this too may be impaired : and in these cases the intellectual faculties may either remain in statu quo or may decline progressively in all respects, reducing the patient to a state of childishness or of pronounced dementia. In other cases, alterations of the temperament may be the principal symptom, the patient generally becoming peevish, whimsical, irritable, or passionate.

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[1] *M. Bernhardt*, Berl. klin. Wochenschr. 1872, No. 32. *H. Cohn*, Ueber Hemiopie bei Hirnleiden. Klin. Monatsbl. f. Augenheilkunde, 1874.

Systematic observations are not yet sufficiently numerous to enable us to say under just what conditions, as regards the seat of the hemorrhage and the form of the disease, these mental changes are especially apt to appear. It is possible that the general failure of the mental powers is associated with the secondary cerebral atrophy.

Symptomatology of the Different Lesions with Regard to their Location.

The reason why the different cases of cerebral hemorrhage resemble one another so closely, from a clinical point of view, lies, as remarked above, in the fact that the seat of the lesion is so constantly in the corpus striatum, the thalamus opticus, the nucleus lenticularis, or their immediate neighborhood. We need not adduce evidence to prove that lesions of parts of the brain having other physiological functions, will give rise to symptoms of a different kind. The question is, whether, from the character of the symptoms, it is possible to decide positively as to the position of the hemorrhage. We hear it declared by some observers that this can readily be done ; by others that it is impossible. For ourselves, we regard this localization as possible, though, with our resources, such as they are at present, we need to use great circumspection in drawing our conclusions.

The reason for this lies, however, not in the nature of the case, but in the deficiency of our observations. It is self-evident that we shall not be likely to advance in this direction if we attempt to utilize every possible case of local disease in the brain, irrespective of the fact that, through pressure, circulatory disturbances or inflammation, the surrounding tissues may have been involved to a greater or less extent, or that the morbid changes may be progressing, or the lesions very widespread. If, on the contrary, we draw our arguments from cases in which the disease (1) has come to a stand-still, (2) is limited in extent, and (3) exerts no influence on surrounding parts, we shall gradually become able to make an exact diagnosis in point of localization. These requirements are fulfilled in the case of no cerebral disease so perfectly as in certain forms of extravasation, not even in the softening from thrombosis. We shall seek in what follows to sketch in outline the rules for localizing the cerebral hemorrhages, limiting ourselves, of course, to the case of hemorrhage alone, and indeed to hemorrhages limited to certain parts of the brain, although what we say will be applicable, to some extent, to cases of thrombosis or embolism. In spite of the immense number of observations existing in the literature of this subject, there are relatively few which can be turned to account in this direction, and in many of these, furthermore, the reports of clinical symptoms are deficient in details which are essential for our investigations. For obvious reasons, we are unable to utilize results of physiological experiments, except to a limited degree and by way of suggestion.

Above all, we desire to emphasize particularly the fact that we are not justified in trying to localize lesions by the presence of any one—so-called pathognomonic—symptom, but must invariably take into account the aggregate features of the case. We shall be obliged to differentiate, in accordance with the limitations which we have laid down, between the symptoms occurring at the moment of the hemorrhage and those which accompany the chronic stage of the affection.

I. *Pons Varolii.*[1]—Hemorrhages, which, if occurring in the hemispheres, would not cause death, though they might give rise to permanent symptoms, may, in the pons, prove rapidly fatal, on account of the anatomical and physiological importance of this centre; if they are sufficiently small, however, they also may give rise to permanent clinical results, preceded by initial symptoms which may or may not be characterized by apoplexy.

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[1] The literature of this subject, up to 1868, is well given by *O. Larcher*, Pathologie de la Protubérance Annulaire. Paris, 1868. 2. Ed.

Extensive hemorrhages destroy life, almost without exception, in a short time, varying from one-quarter of an hour to several hours. The attack is usually ushered in by premonitory signs; but sometimes the patients fall instantly to the ground, perhaps first uttering a sudden cry (morbus attonitus). Deep coma follows. Sometimes universal epileptiform convulsions come on, often more marked on one side of the body than on the other, and involving the face. These are plainly due to irritation of the "convulsion centre," which, as I have proved, is situated in the pons (Virchow's Archiv, 44 Bd.). In other cases, instead of the clonic convulsions, tonic contraction of the muscles of the neck and body occurs, only broken now and then by isolated twitchings. In still other cases all convulsive symptoms are wanting. That these differences in the symptoms are associated with varying positions of the hemorrhage, is very probable, but at present not to be stated positively. We need mention only one fact, namely, that in the great majority of cases the breaking through of the extravasated blood into the fourth ventricle is attended with convulsions; but even under these circumstances it is not true that this symptom is always present, any more than that this particular lesion is the *conditio sine qua non* of its occurrence.

The convulsive movements may last until death, or they may give place to a general muscular relaxation and paralysis of all four extremities. The pupils are usually much contracted, and react but little under the stimulus of light (sometimes, on the contrary, they react well, as Juedell[1] has seen, and I myself), and in this respect hemorrhage into the pons may simulate, clinically, opium poisoning, so that it is sometimes difficult to distinguish between them. On the other hand, well-marked mydriasis has sometimes been seen in uncomplicated cases of hemorrhage into the pons. Finally, pronounced disturbances of respiration are sometimes met with, such as marked stertor, cyanosis, intermittent breathing, and occasionally excessive dyspnœa. All the other symptoms in these cases (and even some of those just specified) may follow hemorrhages in other parts of the brain as well as in the pons.

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[1] Berl. klin. Wochenschr. 1872. No. 24.

With hemorrhages of smaller amount, which are not immediately fatal, the later clinical symptoms may differ exceedingly, according to the precise size and position of the extravasation. The sum of our observations is as yet too small to admit of the establishment of any definite rules with regard to these points, even to the same degree as in other parts of the brain; for we have no right to draw conclusions from cases of recent extravasation which prove rapidly fatal, as is so often done—referring, for instance, the complete paralysis of one-half of the body to the influence of a circumscribed lesion discovered after death—since the symptoms may have been due, in part, to the mechanical action which is common to all hemorrhages, whether here or elsewhere (vide above), and it happens but rarely that we meet with hemorrhages of long standing in the pons.

Motor paralysis is always present in these cases. In view of certain isolated instances of diseases of other kinds in the pons (tumors), we might be tempted to entertain a contrary opinion; yet, so far as we know, this symptom has never been found wanting in case of hemorrhage. At the same time, the distribution and character of the paralysis may differ widely in different cases. The varieties, as at present recorded, are:

1. *General paralysis* of all four extremities, or paraplegia, paralysis of both legs; both forms only in cases that proved rapidly fatal, and where the hemorrhage was extensive, or where it encroached considerably on the parts on both sides of the median line.

2. *Total paralysis of one-half of the body* (that opposite to the lesion), including the extremities, muscles of respiration (in a case observed by myself), the facial and hypoglossal nerves; less often the abducens, the trochlearis, the motor portion of the trigeminus and the motor oculi. Sometimes the leg is abso-

lutely paralyzed, the arm but little (Hughlings Jackson); at other times the reverse is the case (Juedell; two observations by myself). It is believed, by Gubler and Brown-Séquard, that in those cases where the extremities and muscles of the face are affected together, on the side opposite the lesion, the latter is to be sought in the *upper part of the pons* near the crus cerebri; and this has generally been found to be the fact. Leyden[1] has designated the disturbance of speech which is *characteristic of* lesions of the pons and the medulla oblongata, as *anarthria*, the enunciation being more or less inarticulate. This condition, which is due to a paresis of the hypoglossal nerve, is to be distinguished from aphasia (vide the chapter on this subject).

3. In the two cases already mentioned as observed by myself, the right arm was totally paralyzed, the leg paretic; in both the facial muscles were unaffected; in one of them *anarthria* was present; in the other not; in one the sensibility was disturbed over the paralyzed parts; in the other it was intact. In both cases the lesion was seated on the left side of the pons, in the upper part, very near the crus cerebri, close to the middle line; a similar state of things was present in an analogous case of Juedell's. These, together with other observations, prove, therefore, that *paralysis of the extremities may occur without that of the facial or any other of the cranial nerves.*

4. Paralysis affecting at the same time the extremities on one side of the body, and the face on the other,—namely, on that of the lesion,—is very characteristic of affections of the pons (hemiplegia alternans). Here the lesion is situated in the *lower part of the pons near the medulla oblongata.*

The clinical result is explained on the assumption that the nerve fibres coming from the extremities are severed after (*i. e.*, above) their decussation, and those connecting the facial nerve with the cerebrum, before (*i. e.*, below) their decussation in the raphe pontis. *From an anatomical standpoint, however, this explanation is not quite satisfactory.* The accompanying diagram will give an idea of the anatomical conditions which are involved:

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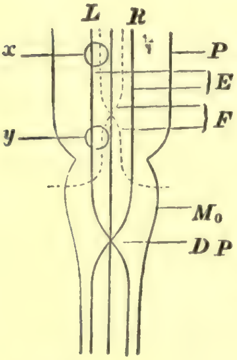
[1] Berl. klin. Wochenschr. 1867. No. 7, et seq.

Lesion *x*: paralysis of right extremities and right side of face.

Lesion *y*: paralysis of right extremities and left side of face.

It was stated above that, in this variety of facial paralysis of cerebral origin, all the branches of the facial nerve, even those supplying the frontalis, corrugator supercilii, and orbicularis palpebrarum, are involved, just as in the peripheral form of this affection; whereas, when the lesion is situated in the upper part of the pons, at least in the cases which I have observed, these branches escape. (For the character of the electrical reaction in these cases, vide above.) Together with the facial nerve the abducens of the same side, *i. e.*, of the side opposite to that of the paralyzed extremities, is often affected in a similar manner.



L = left.
R = right.
P = Pons.
Mo = Medulla oblongata.
DP = Decussatio pyramidum.
E = Nerve fibres for the extremities.
F = Fibres destined for the facial nerve.
x = Lesion in the upper part of the pons.
y = Lesion in the lower part of the pons.

5. *In very rare cases paralysis of the extremities on one side* has been found conjoined with paralysis of *both facial nerves*, although the lesion has been reported to be confined to one side. No satisfactory explanation of this occurrence has yet been offered.

6. Finally, the reverse condition to that mentioned under "three" is to be noted, though it occurs but rarely—*i. e.*, paralysis affecting only the *cranial nerves* and not the extremities. In this case, again, the facial and hypoglossal nerves are most apt to suffer; next, some branches of the motor oculi; seldom all. Under these circumstances it is probable that the longitudinal columns, the prolongation of the crura cerebri, are not involved, but only the fibræ arciformes. It may happen that the lesion is confined to the nuclei of the facial, as in a case reported by Benedict. The peculiar reactions to electricity present in such cases have already been spoken of.

Anæsthesia has the reputation of being a characteristic symptom in cases of lesion in the pons; it is, however, by no means of constant occurrence, and varies, both with regard to intensity and distribution, and also in respect to its relation to motor paralysis. According to a number, both of original and collected observations (cases of extravasation and localized softenings), the following are the forms under which this symptom occurs:

1. Extreme anæsthesia of the side of the body opposite the lesion, associated with paralysis.

2. Slight anæsthesia accompanying complete paralysis.

3. In some cases a greater or less degree of even complete paralysis may occur without any disturbance of sensibility, for the anæsthesia also may affect the extremities upon the side opposite to the lesion and the face on the same side with it (alternate form). According to Hughlings Jackson, the alternate motor paralysis is always present in these cases; but in a case, reported by Leyden, this, though originally present, had disappeared. It seems unquestionable, that loss of sensibility may occur unaccompanied with any disturbance of motion, in view of what has been observed in connection with other localized diseases of the pons; yet we are not aware that this has actually been observed in any case of hemorrhage. It may happen, as in Leyden's case just referred to, that the motor paralysis disappears, leaving only the anæsthesia.

We are far from having a sufficient number of observations to enable us to say exactly what the seat of the lesions must be by which these various results are produced. The fact that the auditory, the trophic, and the vaso-motor nerves are affected, which may or may not be the case, is in no way pathognomonic of lesions in the pons. Larcher calls attention to the fact that the mental functions rarely suffer; but the reported cases of hemorrhages into the pons, where the symptoms have become chronic, are too few in number to justify any absolute statement as to this point.

II. *Medulla oblongata.*—Hemorrhages in this region, if of any size, destroy life in a similar manner to that described in speaking of extravasations into the pons, for in such cases both organs are usually simultaneously affected. A few symptoms may be added to the list, that are sometimes observed, and indeed often ascribed to affections of the pons, though in fact they are only seen when the medulla is likewise involved. Such are simple diabetes insipidus, diabetes mellitus, and albuminuria, which have been clearly proved, by Bernhardt's physiological observations, to be intimately associated with lesions in the fourth ventricle and medulla oblongata.

The reason that these and certain other symptoms are so much more common, as a consequence of chronic diseases in this region—especially the so-called progressive glosso-labio-pharyngeal paralysis—than after hemorrhage, is evidently that the lesions of this latter kind are rarely sufficiently circumscribed in extent. Isolated cases are reported, to be sure, where the symptoms pointed to the existence of an extravasation of ancient date in the medulla oblongata; but the autopsies necessary to confirm the diagnosis are wanting. The few cases that admit of being utilized in this direction present symptoms which are analogous to those seen with other diseases of the medulla—as, for example, paralysis of the tongue (Hughlings Jackson and Lockhart Clarke).

III. *The pedunculus cerebri* is often involved in connection with other parts in the injuries done by hemorrhages in its neighborhood; but it is unusual to find lesions by which it alone is affected. Since conductors for motor, sensitive, and vaso-motor impressions (Budge, Afanasieff) are here collected into one mass, the lesions of the pedunculus may entail disturbances of all these different functions. An almost typical case of this kind, is one reported by Weber,[1] where the nerves supplying the muscles of the extremities, the trunk, and the face, were affected, and also, it seems, the hypoglossal and glosso-pharyngeal nerves, and, moreover, the sensitive and vaso-motor nerves, all on the opposite side of the body from the lesion. It is plain that it must depend upon the extent and position of the lesion, whether all these tracts are affected, and how severely. In the case reported by Weber, the paralysis of motion in the extremities was complete, that of sensation only partial: the extravasation lay in the inner median portion of the pedunculus. This would harmonize with the statement of Meynert[2] and Huguénin,[3] that the centrifugal

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[1] Med.-Chir. Transact. 1863.

[2] Vide, for example, Arch. f. Psychiat. IV. Bd.

[3] Allg. Path. d. Krankh. d. Nervensystems.

(motor) tracts occupy rather the inner half of the pes cruris cerebri, the centripetal (sensitive) the outer portion. Finally, Weber's case presented still another symptom, which, though not always present, is very characteristic of affections of the pedunculus; this is a paralysis of the motor-oculi on the side of the lesion, *i. e.*, *on the side opposite to that of the paralyzed extremities.* As remarked above, this occurs when the trunk of the motor-oculi, in its passage along the inner side of the pedunculus, is involved in the injury of that part. It must, therefore, be absent in the cases where the seat of the lesion is in the midst, or towards the outer part, of the crus cerebri, as in a case reported by Andral. Even when this symptom is present, all the fibres of the motor-oculi are not necessarily affected; but those supplying the levator palpebræ and the iris never escape. Hughlings Jackson calls attention to the fact, that the presence of this variety of crossed paralysis can only be fairly looked upon as indicative of a lesion in the pedunculus, when both sets of symptoms have appeared simultaneously; where this is not the case, this condition may have been brought about by a number of separate lesions, of syphilitic nature, for example.

Lesions in the main cerebral ganglia, from which spring the motor tracts of the crura cerebri, the *nucleus lenticularis* and the *corpus striatum*[1]—inasmuch as they are the most common of all —give rise to the most familiar group of symptoms, of which the outlines have already been sketched. Whichever of the two bodies is attacked, the premonitory signs, the symptoms which accompany the hemorrhage, and those which immediately follow it, may be identical, though it should be added that in a great majority of cases both organs are involved together in the injury. The question remains, what symptoms there are in the chronic stage which point to a lesion limited to one or the other of these parts. The results of my experiments with rabbits would lead us to expect marked clinical differences between these sets of cases.[2] Nevertheless, at the bedside such differences are rarely to be distinguished, and then only to a limited degree.

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[1] Or nucleus caudatus. Both these bodies are usually included, in English, under the name of the corpus striatum.—TRANS.

[2] Virchow's Archiv. 60. Bd.

as will be seen from the following statement, based on cases in which the nucleus lenticularis or nucleus caudatus had at a former time been the seat of hemorrhage limited to that part.

IV. *Nucleus lenticularis.*—Every lesion in this ganglion, unless it is excessively small, causes motor paralysis of the opposite extremities and opposide side of the face. This rule can be regarded as invariable, and it conforms to an unusual degree both with the results of Meynert's anatomical investigations and with those of my experiments, which prove that the so-called psycho-motor nerve-tracts, those which conduct the voluntary impulses downward from the cortex cerebri, traverse in their course the nucleus lenticularis. The paralysis is generally severe and permanent, and may also, in the rather exceptional cases where the lesion is very small, be incomplete in character (paresis). The facial paralysis is of the usual type, *i. e.*, the upper fibres of the nerve escape unaffected. We have pointed out, above, the fact, that in cases of hemiplegia the muscles of the trunk, especially the auxiliary muscles of respiration, are more frequently involved in the paralysis than is generally believed; and in our opinion this is especially the case where the nucleus lenticularis is the seat of the lesion (even in my experiments I found that the muscles of the trunk were almost always affected under these circumstances).[1] The hypoglossus is usually affected only at the outset, as is shown by the disturbances of articulation mentioned above, which disappear after a few days—at least, after the lapse of that time, the speech becomes perfectly normal. The evidences of affection of the motor-oculi, supposing them to have been present at all, disappear again very soon.

Further, another rule may be laid down with regard to the symptoms of lesions in the nucleus lenticularis, which is as invariable as that concerning the motor paralysis, viz., when the lesion is confined to this ganglion no disturbances of sensibility ever result.

They may be present, to be sure, during the first few days; but they are then due, as indicated above, to the initial effect of the hemorrhage as such. The number of our reliable observations is not sufficiently great to enable us to say whether or not vaso-motor or trophic disturbances ever occur.

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[1] Vide Virchow's Archiv. 57. Bd.

When the tract of nerve-fibres passing along the base of the nucleus lenticularis, designated as the *ansa peduncularis* (Hirnschenkelschlinge, Gratiolet, Meynert), is involved in the lesion, the character of the symptoms is somewhat different from that described. The most noticeable difference seems to be that in this case the fibres of the facial nerve supplying the frontalis and the orbicularis palpebrarum, which otherwise escape, are paralyzed like the rest, as in an instance reported by Huguénin.[1] The above-cited case of Chvostek's perhaps belongs in this category. These observations establish the point, of great diagnostic importance, that complete facial paralysis, even if unquestionably of central origin, is not necessarily due to an affection of the pons Varolii.

V. *Lesions of the corpus striatum* also induce, almost without exception, a motor paralysis of the opposite side of the body, which is generally most strongly marked in the upper extremity, sometimes, however in the lower;[2] the facial nerve is likewise involved.

Cases have been reported, however, where lesions in the corpus striatum have been followed by paralysis limited even at the outset to one arm or one leg, or even to the face. It is evident that when this occurs the injury must be confined to some special part of the corpus striatum; but we possess as yet no facts which can guide us in this localization. According to Romberg, a permanent loss of speech accompanying hemiplegia has occurred as a result of a lesion limited (apparently) to the corpus striatum.

The disturbances of sensibility are similar to those observed in affections of the nucleus lenticularis, *i. e.*, when the corpus striatum alone is injured, without the surrounding parts, these disturbances are only of temporary duration. It is probable that the corpus striatum encloses vaso-motor tracts, since characteristic vascular disturbances are met with in the class of cases of which we are treating.

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[1] Correspondenzblatt für schweiz. Aerzte. 1872.

[2] The view, once held by Saucerotte, that only the lower extremities are paralyzed in consequence of affections of the corpus striatum, and only the upper extremities in consequence of affections of the thalamus opticus, has been so often and so thoroughly disproved that it deserves mention only for its historical interest.

The clinical features which render a differential diagnosis possible in the rare cases in which a lesion is absolutely limited to the nucleus lenticularis or the corpus striatum, are, at the best, not sufficiently well marked to render it possible to describe them in a hand-book. We intend to refer to them again elsewhere.

It is still more difficult to define with accuracy the symptoms of lesions in the ganglia of origin of the tegmentum cruris cerebri, the thalamus opticus, and the corp. quad., than of those in the nucleus lenticularis and corpus striatum.

VI. It was formerly assumed as unquestionable that the symptoms of *affections of the thalamus opticus* were essentially identical with those produced by lesions in the corpus striatum; but latterly there has been a strong reaction against this view. So far has this gone, that B. Cohn[1] was induced by his observations to deny that the thalamus opticus stands in any relation to the motor functions of the extremities; and, later, Meynert[2] declared that isolated lesions of this ganglion do not give rise to hemiplegia, and that in the cases where this has been reported as having occurred, a more careful examination would have discovered that the capsula interna, which embraces the outer surface of the thalamus opticus, was involved with it in the injury. For myself, I am entirely of the same opinion[3]—believing that lesions, of which the thalamus opticus is the exclusive seat, are not followed by motor paralysis. Experimental and careful clinical investigations unite in establishing this conclusion.

The greater part of the cases ordinarily to be observed and reported in the literature of the subject cannot be adduced as evidence against this view, since it happens only very rarely that the lesion which injures the thalamus opticus does not involve the surrounding parts, at least if we speak with the strictness which we now know to be necessary. On the other hand, it will be found—if only those cases are taken into consideration in which the affection was confined absolutely within the limits of this ganglion—that no loss of voluntary power is reported as having occurred. It is manifestly impossible to refer to all these cases in detail. Only in one well-known instance, contributed by Andral,[1] does this rule seem to have been broken through; in this case paralysis of the left side of the body with paresis of the left half of the face was observed during life, and, post-mortem, a cavity of the size of a large cherry was found in the interior of the right thalamus. Nevertheless, in view of the fact that at that period but little was known in regard to the exact localization of cerebral lesions, the question may fairly be raised, whether the inner capsule might not have been involved. In face of these considerations, we must, in our opinion, abandon the dictum, hitherto universally accepted, that the affections of the corpus striatum and those of the thalamus opticus are of equal significance for the production of motor paralysis.[2] It may be also regarded as demonstrated that lesions in the interior of the thalamus opticus cause no disturbance of the sensibility.

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[1] *B. Cohn*, Klinik der embolischen Gefässkrankheiten. 1860, S. 402.

[2] In various places, *e. g.*, in the Arch f. Psychiatrie u. Nervenkrankh. IV. Bd.

[3] Vide my experiments; Virchow's Archiv.

We would refer again, in this connection, to a point already touched upon under the head of Disturbances of Sensibility. It was then pointed out that, according to the observations of Türck and Rosenthal, lesions in certain specified regions caused permanent and *marked anæsthesia;* of the various parts which were found to be injured in these cases, the posterior segment of the inner capsule and the adjacent portion of the corona radiata are perhaps the ones of which the destruction is to be considered *essential* for the production of this result.

VII. Hemorrhage into *the lateral ventricles* is usually followed by deep coma, and, after a short interval, by death. The majority of patients die in the course of the first or second day, though they sometimes live until the sixth day, and in rare instances longer. The number of those that escape with life is exceedingly small; though this does happen from time to time, as has been shown by Rokitansky, and confirmed by others (*e. g.*, Charcot, Leçons sur les Maladies des Vieillards). The occurrence of the fatal result, as well as of the deep coma, in these cases, is explained by the fact that the amount of the extravasated blood is very great, so great that it may not only fill one of the ventricles, but, after destroying the corpus callosum and the fornix, damage the opposite side of the brain. It occasionally happens that a person is attacked with hemiplegia (with or without apoplectic symptoms), recovers from his coma—supposing this to have been present—and then, after a few hours, becomes again comatose, and this time does not recover; and that, at the autopsy, a lesion is found in the thalamus opticus or corpus striatum, evidently the cause of the hemiplegia, and an extravasation of blood into the lateral ventricle, which probably occurred later.

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[1] Clin. Méd. Vol. 11.

[2] Vide, in this connection, the statistics collected by Rochoux, according to which, out of seventy cases of hemorrhage the corpus striatum was affected forty-three times, and the thalamus opticus only four or five times.

Attention has already been called to the statement of Durand-Fardel, that hemorrhage into the ventricles is immediately followed by spasmodic contraction of the (paralyzed) extremities on the opposite side, which may either be temporary or may persist until death. No explanation of this occurrence is to be given. This contraction often fails to occur in these cases; and, on the other hand, it sometimes, though rarely, attends extravasations which do not break through into the ventricles. Finally, these extensive ventricular hemorrhages are sometimes followed, besides the contractures, by convulsive movements of greater or less severity. (Vide the observations of Hitz, quoted above.)

VIII. The *corpora quadrigemina* are never attacked alone by hemorrhage, or at least we can find no report of such a case. We have, therefore, no symptoms to describe as characteristic of this lesion. To judge from analogy, and from the results of physiological investigation, we should infer the presence of an affection of the corpora quadrigemina in connection with that of other parts, in case (after the return of consciousness) amaurosis and immobility of the pupil were present—supposing, of course, the existence of other lesions at the base of the brain, or within the eye itself, to have been excluded.

IX. *Cerebral lobes.*—Lesions within these organs are only with difficulty to be recognized, because, according to their seat, they sometimes give rise to symptoms which are identical with those due to affections of other parts of the brain (especially the nucleus lenticularis and the corpus striatum); while in other cases they are unattended by symptoms of any kind.

We will first deal with the question, whether characteristic differences exist between the affections of the left and those of the right hemisphere. The existence of such differences has been asserted at various times—recently, among others, by Fleury,[1] who declares that the functional activity of the left hemisphere lies in the direction of "productivity" or "motility," that of the right in the direction of "sensorial receptivity." Clinical observation, however, has not yet confirmed this statement; and the only clinical evidence for such functional differences is that afforded by the history of aphasia. (Vide the chapter which treats of Disturbances of Speech.) The belief, also, which is still entertained by several observers, that affections of the occipital lobes are especially prone to cause mental disturbances, has but little probability in its favor.

The study of the exact localization of lesions in the hemispheres is as yet beset with especial difficulties, from the fact that none of the accounts of recorded cases, except the most recent, are able to give us the least assistance in this respect. The older observers spoke only of lesions in the "anterior," "middle," or "posterior" lobes, without pretending to specify exactly which of the convolutions was the seat of the injury. Our knowledge with regard to this part of the subject may be comprised in a few general statements.

a. *Cortex Cerebri.*[2]

The study of the circumscribed lesions of the cortex has become a matter of much greater importance than before, since the discovery of the facts that the surface of the brain is susceptible to electrical and mechanical excitation, and that injuries inflicted upon it at certain definite parts may give rise to paralysis (Hitzig and Fritsch, myself, Ferrier). Unfortunately, the results of experiments upon animals, as respects the exact localization of lesions, cannot be transferred directly to the case of man, except that we have reason to believe, through an important experiment of Hitzig's[1] upon an ape, that the motor centres for the nerves of the extremities and the cranial nerves lie in the gyrus præcentralis, or centralis anterior (Huschke, Ecker); in other words, that affections of the cortical substance of this convolution may produce motor paralysis. This observation is in harmony with the teachings of such clinical facts, observed with the necessary care, as are at our command. Of these is to be mentioned an instructive case reported by Hitzig[2] (the evidence afforded by an analogous case of Wernher's[3] does not seem to be convincing; nor does that which is to be drawn from the rather complicated cases of Löchner,[4] Theodore Simon,[5] Hughlings Jackson[6]), and several others contributed by Bernhardt,[7] although according to these latter observations the motor lesion in question does not seem to be confined to the anterior central convolution, but to include as well all the parts bordering on the fissure of Rolando. As negative evidence, it may be mentioned that I have seen cases where circumscribed capillary apoplexies were found in the cortical substance of other convolutions, which had not given rise to paralysis. Lesions of the cortex *may* then give rise to motor paralyses. An observation of Rosenstein's[8] has shown that extravasations into the third frontal convolution may be so small as not to cause any result except aphasia (the whole subject of aphasia will be treated of elsewhere). We have as yet no reliable pathological observations that determine whether lesions of the cortex give rise to disturbances affecting the sensory or to those affecting the vaso-motor functions.

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[1] *Fleury*, Du Dynamisme comparé des Hémisphères cérébraux chez l'Homme. Paris, 1873.

[2] I was unfortunately prevented, by a sudden and severe illness, from taking into consideration, in this connection, the results of a number of recent investigations by Samt and others, which appeared as this work was about to go to press. I regret this all the more from the fact that this chapter was in special need of revision. As it appears, the text contains only a partial and fragmentary discussion of the subject.

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[1] Berlin. klin. Wochenschrift, 1874.

[2] Archiv f. Psych. u. Nervenkrankh. III. Bd.

[3] Virchow's Archiv.

[4] Allg. Zeitschr. f. Psychiatrie. 30. Bd. S. 635, et seq.

[5] Berl. klin. Wochenschr. 1873. Nos. 4 and 5.

[6] Various scattered papers.

[7] Arch. f. Psych. u. Nervenkrankh. 4. Bd.

[8] Berl. klin. Wochenschrift. 1868.

As for extensive hemorrhages into the cortex (which, it may be said, usually take place on the convexity, and at several different points at the same time), since they commonly involve the pia mater to a considerable extent, we shall speak of the symptoms to which they give rise in connection with the subject of Meningeal Apoplexy. We have only one further remark to make, namely, that hemorrhage into the cortex, as we know from experience, if it does not prove fatal, causes a greater impairment of the mental functions than when it occurs in other parts of the brain.

b. *Medullary Substance of the Hemispheres.*

Clinical observations have sufficiently proved that hemorrhage into the medullary substance of the hemispheres may be wholly unattended by symptoms. Hemorrhagic cysts are sometimes found accidentally at the post-mortem examinations of persons who, during life, had presented no evidence of cerebral disease whatsoever; in other cases an apoplectic attack may have occurred, from which a complete recovery had taken place; in still other cases, where the lesion is of the kind described, we meet with the usual evidences of hemorrhage, attended or not by apoplectic symptoms, and followed by disorders identical with those which follow lesions of the corpus striatum, such as paralysis of the opposite half of the face and opposite extremities, or of one leg, or one arm alone, so that a differential diagnosis between the two pathological conditions is impossible. Finally, symptoms of hemiplegia may be present at first, and afterwards disappear. This latter tendency—which seems to be decidedly oftener met with in connection with hemorrhages into the medullary substance than with hemorrhage into the motor ganglia—perhaps deserves more fully than any other symptom to be considered as characteristic of the presence of the lesion under consideration.

Besides the motor paralysis, we sometimes meet in these cases with vaso-motor disturbances and alterations of sensibility. It has already been stated that the occurrence of the extravasation sometimes, though rarely, gives rise to contractures.

Without doubt all these differences in character, between the different sets of symptoms, especially the motor symptoms, point to differences in the seat of the lesion within the medullary substance. We are unable to make any more precise statements in regard to this point; this only is to be regarded as certain, that paralysis may result from lesions of the medullary substance of either one of the lobes. This fact agrees very well with the results of my experiments (l. c.), which have shown that injury of the white substance of the brain in a variety of places may result in paralysis (which in case of rabbits is of temporary duration). Symptoms due to affections of the so-called centrum semi-ovale Vieussenii bear the greatest resemblance to those produced by disease in the corpus striatum. It *appears*—for it can by no means be regarded as proved—as if anæsthesia were more likely to occur when the occipital lobe or the posterior portion of the parietal lobe is the seat of the hemorrhage.

X. *Cerebellum.*—The establishment of the diagnosis of hemorrhage into the cerebellum is beset with even greater difficulties than that of hemorrhage in the parts hitherto considered. As in all the other regions of the brain, so here also, the attack may or may not be attended by apoplectic symptoms. The view maintained by some observers (for example, Remak),[1] that the occurrence of cerebellar hemorrhage is marked, as a rule, by vomiting (and irregularity of the pulse), is disputed by others (Shearer, Leven, and Ollivier).[2] Although it may be regarded as probable that, on account of the nearness of the medulla oblongata, this symptom is more likely to occur or to hold a prominent place in these cases than in others, yet vomiting certainly may occur in connection with extravasation in any part of the brain, and cannot, therefore, be considered as characteristic of hemorrhage into the cerebellum, under which circumstances indeed it is often wanting.

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[1] Berl. klin. Wochenschrift. 1865.

[2] Arch. Génér. 1862, Nov., Dec. 1863, Janvier.

It has sometimes been found that after the immediate effects of the attack have passed away, no evidences whatever of disease have remained behind, and yet the post-mortem examination has revealed the presence of a hemorrhagic cyst of greater or less size. This fact places it beyond question that motor paralysis is by no means so necessary a symptom of cerebellar hemorrhage as it is of lesions in the nucleus lenticularis; for it cannot be said, from the facts now at our disposal, that the occurrence or non-occurrence of paralytic symptoms is something which, in this case, is determined by the seat of the lesion, since lesions, having apparently the same seat, have sometimes been followed by such symptoms, sometimes not. Such being the case, the much disputed question, whether or not the cerebellar hemiplegia affects the side of the body opposite to the lesion or the side corresponding to that of the lesion, is of less importance than at first appears. Perhaps Felix Niemeyer takes the most tenable position, when he affirms that the paralyses which attend affections of the cerebellum are not the direct result of the injury to that organ, but of the action of the lesion upon other parts of the nervous apparatus, so that it depends upon just what parts it is that are thus acted upon, whether the same side of the body becomes the seat of the paralysis, or, as is usually the case, the opposite side. The final conclusion in regard to the whole matter has not yet been reached.

It is doubtful whether disturbances of the co-ordination without paralysis (ataxie cérébelleuse) have ever been observed[1] in connection with simple lesions of long standing, which do not exert mechanical pressure on the surrounding parts, in spite of the fact that the physiologists believe the cerebellum to be a centre for muscular co-ordination; such symptoms, however, if they ever should occur, would be of great diagnostic importance. Where loss of co-ordination of this kind is present, the patient is able to move his extremities perfectly well while in bed, but cannot stand, or certainly not walk alone, on account of severe ataxia.

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[1] Even in the much quoted case of Hérard (Union Med., 1860), the description of the post-mortem appearances shows that the lesion must have exercised considerable diffused pressure.

No reliable cases have been as yet observed where disturbances of sensibility, especially of the nature of anæsthesia, have attended uncomplicated cerebellar hemorrhages of long standing (for the action of recent hemorrhages does not concern us in this connection[1]). On the other hand, amblyopia and amaurosis with mydriasis certainly occur in these cases. The facts at present before us oblige us to conclude, however, that these affections of the optic apparatus are not due to the cerebellar lesion as such, but indicate that the neighboring corpora quadrigemina are also involved.

Finally, certain "compelled movements" (Zwangsbewegungen) have been regarded as characteristic symptoms. In this category belongs especially a peculiar deviation of the eyes, not of that kind which was spoken of above, but such that one eye is rolled upward and outward, the other downward and inward. This symptom seems, in fact, to occur occasionally in cases of acute cerebellar hemorrhage; but whether it is a result of the affection of the cerebellum itself, and if so, how it is produced, are facts which have not yet been ascertained. The other recognized forms of the "compelled movements," especially movements of the body backwards, and the so-called circus-movements, have not yet been observed as a certain consequence of hemorrhage. This is, however, not the place to enter into a lengthy discussion of the subject. Furthermore, the phenomena of this class are not to be laid to the account of disorders of the cerebellum as such, since they attend hemorrhages into, and other affections of, that organ only when at the same time the

XI. *Crura cerebelli* are involved in the lesion. Curschmann[2] has recently reported a rare but important case, in which capillary hemorrhages occurred within a circumscribed spot at the point of junction of the right crus cerebelli with the cerebellum. The patient had been observed to assume throughout her sickness a "compelled position" (Zwangslager) on the right side, the side of the lesion, to which also she instantly returned from any other position in which she was placed. The above-mentioned abnormal position of the eyes was not present. As regards the other symptoms in the case, it was difficult to determine them with certainty, on account of the presence of coma (meningitis basilaris); but there were no evident signs of paralysis. A case analogous in every respect, except that here the peculiar deviation of the eyes was present, has been reported by Nonat.[1] These observations, although not numerous, place it beyond question, especially when the negative effect of so many lesions of the cerebellum is taken into account, that the "compelled positions" and "compelled movements" are to be referred, as stated, not to an affection of that organ itself, but to an injury of the crura cerebelli. The results of physiological investigation are, as is known, entirely in harmony with this supposition.

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[1] The statement made by Gall, that hemorrhages into the cerebellum induce disturbance of the sexual appetite, or are accompanied by erections, has been sufficiently criticised by Longet.

[2] Deutsch. Arch. f. klin. Med. 12. Bd.

To sum up the conclusions which in the foregoing we have, though hastily, deduced from the evidence before us, as to the possibility of localizing hemorrhagic lesions, we find that this can be done with proximate certainty, *in the presence of certain definite conditions*, only for lesions in: 1, the pons; 2, the pedunculus cerebri; 3, the nucleus lenticularis; 4, the crura cerebelli; and, 5, if anæsthesia is the only or prominent symptom, for a certain part of the brain which has been specified above. Furthermore, while we are unable to refer lesions to these parts with anything more than *approximate* certainty, the utmost that we can do for the other regions of the brain, even by dint of the most careful consideration of all the features of the case, is to establish with greater or less certainty a "presumptive diagnosis" (Wahrscheinlichkeitsdiagnose), and often not even this, especially in cases of large and extensive hemorrhage.

Clinical History.

We have spoken in the foregoing of two modes in which cases of cerebral hemorrhage may terminate, viz., by death, following closely upon the initial attack, or by inducing certain symptoms which persist during the remainder of life. We must now pass briefly over the same ground a second time.

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[1] Comptes Rendus. 1861.

It is by no means rare for death to occur in connection with the attack itself, although we are unable to say in just what proportion of cases this occurs. In other cases the patients survive the—so to speak—primary coma, become partially restored to consciousness, remaining for a few hours or days with their minds partially clear, and then fall off again into an unconscious state. This is due to the occurrence of a new hemorrhage, either in another place, or, what is more common, in the same place with the first; and such cases almost invariably end fatally.

Supposing the patient to have outlived the attack itself, he may be again in danger of death during the period of reactive inflammation.

The fatal result, under these circumstances, is probably due to encephalitis (exactly how it acts it is not known), though fatal affections of the lungs also sometimes carry off the patient during this stage of the disease.

Even with the close of this period the patient has not escaped from all danger. His life is likely to endure for a long time, perhaps ten, or even twenty years; but sooner or later the hemorrhage, in the majority of cases, recurs. It may be again and again recovered from, it is true, but now and then it proves fatal.

This tendency to a recurrence of the hemorrhage indicates that the conditions in which it originated still persist, *i. e.*, that not one only, but a large number of miliary aneurisms had been produced by the original disease of the vascular walls. Of course a hemiplegic patient is liable to be attacked with intercurrent diseases of various kinds, of which pneumonia[1] seems to be the most common.

The often-proposed question, whether it is possible for cerebral hemorrhage to end in complete recovery, should receive, in our opinion, both an affirmative and a negative answer. It is plain that in every case certain changes are produced in the cerebral tissues, which must, to some extent, be permanent; nevertheless, it will depend entirely upon their position whether the symptoms excited by them pass, finally, entirely away or not. This they may do, it is plain, when the cicatrix resulting from the extravasation is so situated as not to cause any disturbance of the cerebral functions by its presence—a rare event at the best, but one seen occasionally, in cases of lesions affecting the medullary substance of the hemispheres.

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[1] Durand-Fardel has compiled tables indicating the relative frequency of occurrence of the above-mentioned complications: the number of cases on which they are based, is, however, so small that the conclusions are of no great value.

There would be as little justice in refusing to consider this result as one of complete recovery, as there would be in doing the same for a case where, for example, a flesh wound on the body had healed, leaving a deep scar, but not impairing the function of any part; in short, the whole discussion is essentially a dispute as to the meaning of the terms employed.

Prognosis.

We need not adduce evidence to show that cerebral hemorrhage is invariably—both in its early and in its later stages—a serious affection. In the preceding pages we have repeatedly pointed out the clinical significance of the various special symptoms (both those accompanying the attack itself and those occurring later), and it is unnecessary to go over precisely the same ground again. One point alone deserves to be dwelt upon; viz., inasmuch as, according to the statistics collected by Durand-Fardel, it seems that hemorrhages which break through into the ventricles and meningeal cavities are of relatively frequent occurrence with patients over sixty years of age, the prognosis at the outset, in cases of this class, would be less favorable than in the case of younger persons. At the same time, it is possible that hemorrhages of a different kind from this, and resulting in cicatrization, may occur, even with patients of this advanced age; and, therefore, unless certain pathognomonic symptoms are present, it would be necessary to await further developments before laying down an absolute prognosis of this sort.

The differential

Diagnosis

of cerebral hemorrhage, which has to deal principally with the means of distinguishing it from thrombosis and embolism, will be treated of in connection with the subject of *softening.*

Treatment.

The treatment of cases of hemorrhage, corresponding to the different stages in the clinical history, may be discussed under the heads of: *a*, prophylaxis; *b*, the treatment of the attack itself; *c*, that of the stage of reactive inflammation; *d*, that of the permanent resulting disturbances.

a. Is it possible to guard against the occurrence of the hemorrhage itself? If the etiological relations of the affection are borne in mind, it will be seen that this question naturally divides itself into two parts, to one of which a negative answer must be given.

Supposing, namely, the view which we have advocated to be correct, viz., that the real cause of the hemorrhage lies in the presence of miliary aneurisms, it is plain that we have no means in our hands to prevent its occurrence, since we are absolutely ignorant of the conditions in which these aneurisms have their origin.

On the other hand, those influences which we called exciting causes also play their part in bringing about the result, and in removing or preventing the occurrence of these, we are able to exert a certain degree of prophylactic influence. Since these influences are identical with those present in the case of cerebral hyperæmia, we may refer the reader, for their consideration, to the chapter upon that subject.

b. The occurrence of the hemorrhage itself, especially if attended with symptoms of apoplexy, gives rise to such a serious group of symptoms that the interference of the physician seems imperatively called for—so much so, indeed, that the tendency often is to interfere too much. An unprejudiced study of the facts has, however, shown that even his most energetic efforts are usually fruitless.

Of all the various methods of treatment, that by *bleeding* has, from the earliest times, been the most conspicuous. This was formerly regarded as so important that venesection of the arm was sometimes considered insufficient, and, instead of it, the jugular vein, and even the temporal artery, were opened; but the reaction from this excess led to the almost complete abandonment of bleeding, as a useless and even injurious measure (consult, for example, Dietl, Trousseau). The true path, which is now followed by the majority of good practitioners, lies evidently between these two extremes.

Before laying down rules for the practical application of this means of treatment, it may be worth while to discuss, from a theoretical standpoint, the question, *what effects have we a right to expect from venesection in cerebral hemorrhage?* The theory, that thereby the absorption of the extravasation is promoted, could hardly find supporters at the present day; we need, therefore, give no space to its discussion. Is it to be supposed, however, that bleeding exerts a hæmostatic action —a belief under which it is often employed? A direct influence of this kind it certainly does not exert. It is well known that where hemorrhage occurs on exposed parts of the body, venesection has a styptic effect only when carried to the point of producing syncope; experience has shown, however, that it cannot safely be used to this extent, especially in case of hemorrhage within the brain. Furthermore, we know, through our anatomical investigations, that the fibrinous clot, which is immediately formed out of the extravasated blood, is the true styptic under these circumstances. On the other hand, it is possible that when the pressure on the arterial system is abnormally great, its diminution should help, somewhat, to restrain the bleeding; too much weight should not, however, be attached to this influence. The suggestion, that the venesection tends to prevent a renewal of the hemorrhage, has as little foundation as the foregoing, and that for the simple reason that such a renewal has been practically found to occur but seldom—supposing the escape of blood to have once fully ceased; nevertheless it must be acknowledged that this event, or the rupture of another aneurism, would be less likely to take place if the arterial tension were diminished.

The chief indication for venesection is, in our own opinion, of a different nature from either of those mentioned. Its value in cases of cerebral hemorrhage lies in the fact that it brings about a diminution of the intracranial pressure (*i. e.*, indirectly, of course, by diminishing the arterial tension), and in this way its influence may be at times of capital importance. In case, namely, the extravasation has caused an increase of the intracranial pressure, and that in consequence of this, together with the *cerebral hyperæmia that accompanies it*, a paralysis of the respiratory, or of the vagus centre is threatened, the rapid reduction of the quantity of the circulating blood—for it is impossible to act directly upon the extravasation—may, by diminishing this pressure, have the effect of actually prolonging life; and this indication can only be fulfilled by venesection.

On the other hand, the fact ought not to be lost sight of that a certain quantity of arterial blood is necessary to the maintenance of the functional activity and excitability of the brain. In seeking, therefore, to diminish the intracranial pressure through venesection, the danger is to be guarded against of carrying this so far as to produce anæmia, a consideration which demands, what experience has already sufficiently taught, that bleeding, if used at all in such a case, should be moderate in amount. It is self-evident that the observance of this rule is all the more important in case the functional efficiency of the heart is already below the normal.

The explanation of the action of venesection, as just described, is in harmony with the observed fact that patients have sometimes been roused by its aid from a state of coma.

A beneficial effect may be looked for from venesection in cases where apoplexy is accompanied by well-marked symptoms of cerebral hyperæmia, such as turgid face, with perhaps distended veins and increased pulsation of the carotids; where the action of the heart is powerful, the radial artery of at least normal tension, the pulse slow or regular, and of normal frequency; where the respiration is uniform, quiet, and of snorting character; when the patient is strong and of not too advanced age.

Supposing these conditions to be present, we regard venesection as indicated, if cerebral pressure, already considerable, has begun to cause paralysis of the vagus centre (rapid pulse) and the respiratory centre (Cheyne-Stokes respiration); at any rate, through the use of this measure, combined with stimulant remedies (vide below), life may still be preserved in a certain proportion of these cases.

The formulating of this positive indication constitutes in itself a sufficient expression on our part, of the opinion, that venesection is out of place in all cases that do not correspond to this description. In many cases it is indeed directly injurious, and may hasten the occurrence of death by weakening the action of the heart. This is to be especially borne in mind in case of decrepid persons, where marked arterio-sclerosis is present (not to speak of valvular diseases of the heart), and where the arterial tension is feeble.

Where venesection is indicated at all, it should be general; local bleeding by leeches, or cupping at the back of the neck, or over the mastoid processes, cannot be substituted for it. In the case of children, to be sure, it is sometimes necessary to make use of these latter means.

Besides and together with venesection, all those agents are to be employed which diminish the cerebral hyperæmia by their "*derivative action*," such as mustard-plasters on the calves of the legs, Junod's boots, stimulating enemata. The mode of action of these remedies has been indicated in the chapter upon Cerebral Hyperæmia. It is self-evident—and the same is true with regard to applications of cold upon the head—that none of these modes of treatment can be substituted for bleeding, when this is really called for, either as regards the rapidity or efficiency of their action; but they may be used to advantage in connection with it in cases where the symptoms are very threatening. In light cases, and when one is in doubt whether or not venesection is demanded, these derivatives may be used in its place. The general rule, enjoining that the patient should lie with the head raised, should be moved slowly and carefully, and should be protected from all influences which might disturb the circulation and respiration, need only be mentioned in passing. It is important also, where the stage of unconsciousness is of long duration, to examine the bladder, and, if necessary, to use the catheter.

It is not in every case, however, as has been already remarked, that the occurrence of the hemorrhage is attended with such symptoms as require, or justify, venesection or derivative treatment. In many cases, indeed, precisely an opposite course must be pursued, namely, one of *stimulation.* The indications for treatment of this kind may lie in symptoms which attend the hemorrhage itself; but it also happens that they may arise under the hands of the physician in consequence of too energetic venesection. A stimulant treatment is called for where paralysis of the respiratory centre is threatened and the heart's action is feeble. The condition of the patient under these circumstances is the following: the countenance is pale and collapsed, or, to say the least, not turgid; the arterial tension is slight, the heart's impulse weak (the frequency of the pulse is of comparatively little consequence, and no great importance is to be attached to the height of the pulse wave, since, in case of cerebral hemorrhage, increased tension, such as may indicate venesection, may accompany a small pulse); the respiration will have begun to be hesitating and intermittent, and to take on the Cheyne-Stokes character.

This group of symptoms may be met with both in the case of persons of advanced years or already feeble, or who are suffering from diseases of the vessels, and in the case of robust individuals where the intracranial pressure is greatly increased. In certain cases of the last-named variety it may be that venesection and stimulant treatment will be indicated together, or one after the other; but this must be a matter to be decided by special consideration in each case.

The stimulant agents are in part such as will, by irritating the nerves of the skin, arouse the depressed activity of the respiratory centre. In this manner act: sprinkling of the skin with cold water; the application of ammonia to the nostril; scrubbing or rubbing the skin; or burning it with drops of hot sealing-wax, and the like; or substances may be given which stimulate directly the heart's action, *i. e.*, maintain and increase the irritability of the cardiac centres—this is perhaps the action of musk (too little employed in such cases)—coffee, wine, and stimulating preparations of ammonia;[1] if the patient cannot swallow, enemata of musk may be used.

The large percentage of deaths from apoplexy proves how fruitless is often the most careful and best-considered treatment; and when it is remembered, furthermore, how frequently patients recover spontaneously from the attack, it seems fair to raise the question whether efforts, such as those described, or medical interference of any kind, are ever of value. It would be difficult to convince a determined skeptic that such is the case; but the affirmative view gains a certain degree of probability through the fact mentioned above, that venesection is often immediately followed by return of consciousness. This being taken into consideration, and in addition the fact that the efficiency of the remedies in question, if it cannot be proved, is not to be disproved, and that without them we have no treatment left, we should, in our opinion, continue to employ them, and that too not simply from a humanitarian regard for the friends of the patient.

c. Supposing the patient to have survived the first storm of the apoplectic attack, the less that is done for him during the first few days the better. He should of course be given complete bodily and mental rest; and all substantial or stimulating diet, especially coffee, wine, etc., should be withheld. The next question is, what shall be done when the febrile symptoms mentioned above become still more strongly marked? It was formerly the practice to turn immediately to the lancet; but this is no longer customary. Experience has taught that venesection is rarely necessary. For the severer cases a local bleeding with leeches or cups is sufficient; yet even this is often superfluous, being only indicated when turgor faciei, severe headache, and considerable fever are present. In other cases the application of cold to the head is all that is necessary.

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[1] The employment of tartar emetic and ipecac is now only a matter of historical interest.

Special attention should be paid to the state of the bowels through the whole of this stage; and especially if there is any febrile reaction, they should be kept free.

If the headache still continues, accompanied by persistent wakefulness or delirium, an opiate or chloral may become necessary. It is doubtful, however, whether any of these measures will invariably control the symptoms in the severest cases.

d. We have next to discuss the therapeutic relations of the permanent symptoms, the paralyses, such as are almost invariably present. In judging of the results of treatment, it should of course be borne in mind that spontaneous improvement of greater or less amount (vide above) often takes place. We cannot then fairly decide as to the value of any particular remedy, unless we wait, before employing it, until three or four months have elapsed.

The efforts of physicians have been directed hitherto towards one of two ends, viz.: it has been their aim either to hasten the *absorption of the extravasated blood*, or to improve in some way the condition of the paralyzed parts. The belief, that we can hasten the absorption of the extravasation by any more special method than by enforcing intelligent hygienic rules, is now abandoned. There is not the least reliable proof that the condition of a hemiplegic patient can be improved by efforts of that nature. Physicians have therefore ceased to employ the various derivative agents, such as leeches, cupping, blistering, the use of irritating ointments, etc., applied about the head for the exclusive purpose of quickening the absorption; and also the inward and outward application of the so-called alteratives and absorbents, of which the preparations of iodine and quicksilver were the chief. These drugs are even capable of doing harm, by interfering with the appetite; and certainly the absorption of an intra-cerebral extravasation has never yet been brought about by iodine, not to speak of the other substances employed for the same purpose. We know no single means which can conduce to this end (possibly with the exception of electricity), and are therefore obliged to rely on the natural reparative processes, confining our efforts to the removing of all influences that might interfere with their action.

In the accomplishment of this latter object, besides the prophylactic means referred to above, reference should be had to the following points: the diet of the patient should be at once nourishing and digestible, while all substances tending to cause flatulence or to excite the action of the heart are to be carefully avoided; he should keep strictly out of the way of excitement, and at the same time should receive the benefit of passive or active exercise and the open air; the bowels should be kept regular.

After the immediate effects of the primary attack have fairly passed away, the bladder usually resumes its normal functions, and the occurrence of bed-sores and pulmonary diseases is also no longer to be feared.

Admitting that there is but little prospect of bettering the condition of the patient by acting directly upon the diseased portion of the brain, it must be acknowledged that not much more is to be hoped from striving after the *second proposed object of medical treatment*, namely, the *improvement of the condition of the paralyzed nerves and muscles.* The important results which have recently been attained in the treatment of other nervous diseases, through the use of electricity, the water cures, etc., are met with only to a very limited extent in cases of hemiplegia from hemorrhage; nevertheless no one who is not wilfully blind to the facts can help acknowledging that there are some cases which we are able to make better, to some extent, by treatment.

The practice, formerly in vogue, of using liniments containing alcohol or other irritating substances, was in a measure justified by the fact that at that time no better treatment was known. No definite reason can be given for believing that these cutaneous irritants have any effect in helping to the restoration of motor power; at the best their usefulness must be very limited.

The use of strychnia, also formerly such a favorite remedy in cerebral hemorrhage, has been nearly abandoned, since careful investigation has failed to prove its usefulness; if any one desires to try it, as a last resort, he should do so only in chronic, stationary cases, since unpleasant results (severe contractures, fresh hemorrhage) have sometimes followed its use in recent cases.

Electricity stands at the head of the list of agents which are really of value. Both the galvanic and the faradic currents have been used—in later times especially the former, which indeed really seems to be the more useful of the two. We will turn our attention first to the constant galvanic current. This form of electricity is without effect in many cases of hemorrhagic hemiplegia. On the other hand, however, it is certainly sometimes possible, through its use, to improve the condition of the patient to a certain extent even in old cases—the subjects of marked contractures, which have resisted every other kind of treatment. For this reason, and because, if carefully employed, it can do no harm, it is unquestionably worth while to make at least a trial of the constant current in cases of hemiplegia of long standing.

It would be out of place here to discuss in detail the general subject of the application of the constant current, and the theoretical question, upon what its anti-paralytic action depends (it is variously designated, as electro-tonic, invigorating (Heidenhain), centripetal (Remak)), and we must limit ourselves to a few brief and practical statements. The galvanic current may be applied with either of three different objects, viz., for the purpose of acting upon the paralyzed limbs, the cervical sympathetic, or the brain itself. When applied to the brain itself, which is accomplished by conducting it directly through the skull, the electricity is expected to affect the condition of the extravasation itself, tending to cause its absorption in virtue of its catalytic action. It is scarcely doubted by any one at the present day, in view of the various symptoms which are produced by applications of this kind, and of the facts ascertained by direct experiment (Erb), that a portion of the current, under these circumstances, traverses the brain; the proof, however, is entirely wanting for the assumption that the absorption of the extravasation is in any way hastened thereby. At the same time, the statement, first made by Remak, and confirmed later by various observers, has been found to be practically correct, that noticeable improvement, as regards both the paralysis and the contracture, is sometimes brought about by means of this treatment alone. Another method, which aims at stimulating the cervical sympathetic, usually conjoined with a mode of application in which the electricity is made to traverse the head from side to side, rests, like the above, on a purely empirical basis.

In applying galvanism about the head, the well-known precautionary measures laid down in every hand-book of electrotherapeutics are to be borne in mind. The duration of each application is to be short, hardly exceeding three minutes ; the current should be weak—weaker with elderly than with younger persons—such, for example, as is derived from four to twelve Siemens-Halske elements ; the strength of the current should be gradually increased from zero, and gradually diminished ; the electrodes should be gradually removed ; sudden interruptions of the current should be avoided, and, still more, rapid reversals of the current. It seems to be a matter of slight importance whether the electrodes are placed upon the two mastoid processes, or one on the mastoid process and the other on the back of the neck.

As a rule, the peripheral galvanization of the paralyzed nerves is used in connection with applications of the kind described.

For this purpose one of the electrodes (generally at the negative pole) is commonly placed near the plexus to which the affected nerves belong, or over the corresponding part of the vertebral column, the other upon the parts over the trunk of the nerves themselves.

Under these circumstances it is allowable to use stronger currents and for longer periods than under the previous method (up to thirty elements, and during eight minutes) ; and, in order to make the stimulating action of the current as great as possible, its intensity may be alternately increased and diminished, while the circuit is kept closed. This end is accomplished by the use of the so-called labile currents of Remak ; but, while absolute interruptions and reversals of the current, such as excite muscular twitchings, are scarcely ever used in such a case for therapeutic purposes, they may be for purposes of diagnosis.

Still less is to be expected from the use of the faradic than from that of the galvanic current in cases of hemorrhagic paralysis. Duchenne, the most experienced worker in this field, makes very unfavorable statements as to the results which are to be attained in this way, and the experience of almost every other observer coincides, in general, with his. Electrical currents of this kind are never used for the purpose of acting directly upon the brain, but only upon the paralyzed muscles and nerves, and, in cases where contractures are present, upon the antagonists of the contracted muscles.

Nevertheless, it happens not unfrequently that when the contracture is severe, it becomes still more so under the influence of electrization, even though applied to the antagonistic muscles. We repeat that improvement may follow the use of the induced current; but, on the whole, this is less effective than galvanism.

It has become established as a general rule that the use of electricity shall not be begun too early, lest untoward accidents, such as now and then occur spontaneously, may be induced—for example, fresh hemorrhage or cerebral congestion. According to Duchenne, treatment with the induction-current should begin five or six months after the hemorrhagic attack. In light cases it may be used with care at an earlier period; but it must always remain questionable in such cases whether such improvement as may take place should be ascribed to the treatment or to natural causes. The same may be said with regard to the constant current.

Besides electricity, there is really only one agent of any material value in cases of cerebral paralyses, and that is the *use of hydropathic treatment.* At the present day, just as in former times, when the use of electricity was unfamiliar, these unfortunate hemiplegic patients are to be seen making their way to the various water-cures and returning home unimproved; for, of all kinds of paralyses, those of which we are now treating (and perhaps the same may be said of the hysterical variety) are the ones which obtain the least benefit of any from this kind of treatment, which in other forms of paralysis often accomplishes so much. At the same time it cannot, fortunately, be denied that now and then real improvement, increased power over the paralyzed extremities, is brought about by the judicious use of this kind of treatment.

More important than the choice of a special water-cure, of which there are many whose action is essentially the same, is the strict observance of the fundamental rule, that *hemiplegic patients should only use baths of moderate temperature.* It is true that the neglect of this rule is not always immediately harmful in its effects; but it is certainly true that such neglect may result, and frequently has resulted, in causing fresh extravasation. This rule being observed, there seem to be a variety of baths and water-cure establishments which are of equal value. The best known are, among the moderately warm baths, Wildbad, Pfäffers and Ragatz, Landeck (Teplitz, to be used only with great caution), and among the brine baths, Rehme and Nauheim.

Cold-water treatment, if used under skilled supervision, may be of service in isolated cases; as a rule, however, it is not indicated in cases of hemorrhagic paralysis; and the same may be said of sea-bathing.

B.—Meningeal Hemorrhage.

The history of our acquaintance with meningeal hemorrhage is so intimately associated with that of intra-cerebral hemorrhage that in essential respects it will be found to have been already laid down in the foregoing pages; it has, indeed, always been the custom to treat of the two subjects in conjunction. Virchow's investigations as to the relation between hæmatoma of the dura mater and pachymeningitis interna hemorrhagica[1] deserve, however, special mention. Through their means (the observations of Heschl,[2] made independently, are entirely in harmony with the conclusions derived from them) a large group of cases has been removed from the category of the essential and meningeal hemorrhages, and referred to that of pachymeningitis, and thereby the symptomatology of the former affection, previously somewhat complicated, has been greatly simplified. Sperling,[3] to be sure, has, in view of experimental evidence, readopted the earlier view that the hemorrhage is the primary process, and that the formation of the pachymeningitic pseudo-membranes is due to organization of the extravasated material. Before Sperling's view can be accepted as correct, however, his statements must be subjected to further investigation. The requirements of a hand-book oblige us to accept for the time being the conclusions established by Heschl and Virchow. We shall therefore refrain in what follows from speaking of hæmatoma of the dura, leaving it to be discussed among the inflammatory affections of the membranes.

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[1] Verhandl. d. med.-physik. Ges. z. Würtzburg. 1856.

[2] Path. Anatomie. 1855.

[3] Ueber Pachymeningitis hæmorrh. Inaug.-Diss. Königsberg, 1872.

Etiology.

The first variety of meningeal hemorrhage to be mentioned in this connection is one which properly belongs in the department of surgery, viz., that of *traumatic origin.* Direct injuries of the main meningeal arteries, the sinuses, or the vessels of the pia mater, due to broken fragments of bone, may give rise to hemorrhage. It happens exceedingly rarely that the vessels lying near the base of the skull are reached by direct injury, yet such cases have occasionally been reported.

On the other hand, extensive meningeal hemorrhages sometimes take place from the arteries at the base of the skull, from another cause, namely, from the rupture of *aneurisms,* of which they are the seat. The vessels which are most likely to be affected in this way are the basilar and middle cerebral arteries. Hemorrhage may also take place from the veins; the ordinary venous stasis rarely leads to rupture, it is true, but extravasation into the meninges of serious amount is no rare complication of *thrombosis of the sinuses,* especially the superior longitudinal sinus.

In other cases, blood may make its way into the meninges by bursting through from within the substance of the brain, escaping finally either at some part of the convexity or by way of the ventricles.

Meningeal hemorrhages are also observed as a complication of the various so-called *infectious diseases,* and some of the general constitutional affections, such as were specified in the chapter on Intra-cerebral Hemorrhage (vide p. 70, et seq.).

Meningeal hemorrhage, often of considerable amount, is also seen at times in cases of the so-called general progressive paralysis of the insane, originating in the congestion of the vessels of the pia mater.

Ludwig Meyer,[1] for example, discovered this condition post-mortem in ten cases out of one hundred and sixty-eight, and in three it had been so extensive as to cause immediate death.

A special form of this hemorrhage, the so-called *meningeal apoplexy of new-born children*, may occur in consequence of certain influences attending childbirth. When, namely, the bones of the skull are made to lap over each other in the course of severe forceps-extractions, or prolonged and difficult labors of other kinds, with or without contraction of the pelvis, rupture of the vessels of the pia, or, as has been occasionally observed, of the longitudinal sinus may result, which usually ends fatally.

Pathological Anatomy.

The seat of the hemorrhage may vary. In rare cases—for example, when the middle meningeal artery is injured—it occurs between the bone and the dura mater. Besides this, sharp distinctions were formerly drawn between hemorrhages occurring: 1, between the dura and the so-called parietal layer of the arachnoid; 2, in the so-called arachnoid cavity; 3, between the visceral layer of the arachnoid and the pia and within the latter (subarachnoid space). Those of the first category, which were formerly regarded as relatively the most common, are now classed under the head of pachymeningitis interna hemorrhagica, especially since it is no longer believed that any such thing as a parietal layer of the arachnoid exists. Furthermore, the other two varieties often occur conjoined, and, especially from a clinical point of view, it is useless to attempt to classify them separately. The extravasated blood may find its way into the so-called arachnoid cavity, either from without, in consequence of injury of the dura mater; or from within, by bursting through the pia; or, finally, it may escape into it directly from the vessels of the latter membrane.

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[1] Virchow's Archiv. 58. Bd.

According to Virchow, the hemorrhage in apoplexia neonatorum usually takes place into the arachnoid cavity; but Bednar[1] regards this as of relatively infrequent occurrence, believing that the commonest event is a sort of ecchymotic extravasation into the meshes of the pia.

The seat of the hemorrhage is sometimes at the base, sometimes on the convexity, sometimes in both places at once; the latter is more likely to occur when the amount of the extravasation is large, and especially in case it spreads extensively through the so-called arachnoid cavity. Sometimes a single hemisphere is exclusively or pre-eminently affected; at other times nearly the whole surface of the brain becomes covered with quite a thick layer of blood. The quantity of the effused blood also varies: it may be so small as only to occupy a space of the size of a lentil, or may amount to half a litre or more.

When death supervenes in the course of a few hours or days, as is usually the case, the blood is found dark and coagulated; if life endures longer, the surface of the brain becomes infiltrated with serum.

Can meningeal hemorrhages be reabsorbed? If of large amount they probably *prove invariably* fatal; but the presence of pigmented spots on the meninges and the surface of the brain, after death, seems to indicate that the absorption of slight extravasations is a matter of possibility.

The appearances in the brain at large vary according to the cause of the hemorrhage. If it was due to the rupture of an aneurism, the changes, to which the pressure exerted by the latter have given rise, will be present, or the brain substance in its neighborhood will be found to have suffered mechanical injury at the time of the rupture; while, if it perforates the brain, the evidences of that occurrence will be visible. Where the amount of the extravasation has been great, the gyri are found to be somewhat flattened, and the substance of the brain at large pale; though in the cases of apoplexia neonatorum, the brain substance is, to be sure, usually infiltrated with serum, and vascular. It is not uncommon for the blood, if extravasated in large quantities, to make its way into the ventricles; and it may even run down into the central canal of the spinal cord, as happens especially in cases where large collections had formed at the base of the skull and around the pons Varolii.

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[1] Die Krankheiten d. Neugeborenen und Säuglinge. II. Bd. Wien, 1851.

Symptomatology.

It is important, for the sake of clearness of description, to distinguish between hemorrhage occurring in the adult and that occurring in new-born children. Even of the former class of cases, however, there is only one variety of the affection which is attended by definite and regular symptoms. The clinical history of meningeal hemorrhages of traumatic origin is almost invariably complicated by other cerebral symptoms directly traceable to the injury (commotio cerebri), and similar statements may be made with regard to the cases where an intra-cerebral hemorrhage has broken through to the surface of the brain, as well as to those of general paralysis. The least complicated class of cases are those where the rupture of an aneurism is the cause of the hemorrhage, and to this class we shall direct our attention first and principally. The symptoms naturally vary somewhat, according to the quantity of the extravasated blood; but, *as a general rule, the symptoms of meningeal hemorrhage are almost identical with those which characterize the apoplectic stage of extensive intra-cerebral hemorrhages;* in other words, the symptomatology of uncomplicated cases of meningeal hemorrhage is the same with that of the apoplectic attack.[1]

Disregarding the symptoms present before the attack, which are referable to the influence of the aneurism as such, and are described elsewhere, the attack itself may be said to occur without warning, or with only slight premonitory signs, of which the chief are headache, dizziness, and, less often, vomiting, symptoms which no doubt correspond to the commencement of the extravasation, since, as was remarked, the apoplectic symptoms in these cases are closely analogous to those due to intra-cerebral hemorrhage; the description already given of the latter may, then, suffice for both. We need only pay special attention here to a few points of difference between the two sets of cases.

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[1] Vide *Lebert*, Ueber die Aneurysmen d. Gehirnarterien. Berl. klin. Wochenschr. 1866.

The paralysis (occurring in coma) is commonly general, affecting all four extremities uniformly. This happens even when the extravasation is confined mainly to the surface of one hemisphere, evidently on account of the greatly increased, diffused intra-cranial pressure. Only in exceptional instances is hemiplegia met with, and in such cases the facial muscles may be involved in the paralysis. The frequency with which convulsions of epileptic nature occur also serves to distinguish these cases from those of intra-cerebral hemorrhage. Meningeal hemorrhage may arise from the rupture of an aneurism of almost any artery, but it is especially common with those of the middle cerebral and basilar arteries (Lebert). Vomiting is another very common and characteristic symptom. Several observers have also described severe bilateral or unilateral contractures of the extremities.

The serious nature of these meningeal hemorrhages is shown in the fact that they are much more likely to cause death during the stage of coma than are most of the varieties of cerebral hemorrhage; indeed, in speaking of the latter, we pointed out that the fatal result in a large proportion of the cases of apoplexia fulminans was due to the bursting through of the blood into the meninges. According to Lebert's statistics, out of forty-eight cases of rupture of aneurisms death occurred in seventeen within ten hours.

The clinical history is, however, not invariably such as we have just indicated, *i. e.*, characterized by sudden coma, ending fatally within a few hours or days. It has sometimes happened that, after passing through an apoplectic stage of short duration, the patient has again come to himself, and for a period, varying in length between several hours and two days, has complained only of headache, with excitement, or, on the other hand, somnolence, and at last has fallen anew into a state of coma, which this time has proved fatal; such, for example, is the history of a patient of Boudet, cited by Durand-Fardel. In other cases an apoplectic attack is not the first symptom of the affection. The patients complain only of headache, dizziness, weakness, and numbness in the extremities on one or both sides, are mentally dulled, but do not fall into a state of fatal coma for a long time—in two cases of Durand-Fardel, not until after the lapse of a month. In one of these cases, which are described in the original work at length, the amount of the extravasation was, to be sure, not very great, the blood not even covering one entire hemisphere. In cases with this history, where the amount of the extravasation is large, the question might fairly be raised whether it was present in the very beginning. It seems at least to be beyond doubt that it must be believed to have undergone a gradual increase. In the cases, however, where, after an injury to the skull, the patient loses consciousness only for a few moments, then is able to continue his work for some days, and, finally, at the end of that time dies in a state of sopor, as happened in a case reported by Gibson (Edinburgh Med. Journal, 1870), and where, post-mortem, a fracture of the skull is found, together with a rupture of the middle meningeal artery, it is difficult to help acknowledging that the meningeal hemorrhage must have taken place in its entirety at the very outset, or at the least have begun at that time.

It can scarcely be doubted that the symptoms, such as have been described in cases of hemorrhage into the meninges, are due to the great increase in the intracranial pressure, which acts in the manner already described in a former chapter. The experimental investigations, especially those of Leyden and Pagenstecher (ll. cc.), into the effects of increased cerebral pressure, furnish sufficient proof of this view; they found that, with dogs also, extravasation into the meninges (artificially produced by injection of fluid) induces loss of consciousness, general convulsions, stertorous respiration of the Cheyne-Stokes character, etc.

We have no definite description to give of the symptomatology of circumscribed hemorrhages, accurate observations being as yet too few in number. One case of the kind, reported by Durand-Fardel, has already been spoken of.

The meningeal hemorrhages of new-born children, which arise during delivery, were formerly regarded as a fruitful cause of the simulated and real death of the infant. Cruveilhier, for example, refers about one-third of the cases of so-called asphyxia neonatorum to this cause. Since that time there has been a reaction against this view (H. Schwartz,[1] Pernice[2])—Schwartz, especially, seeking to prove that intra-cranial hemorrhage is the cause neither of the simulated nor of the real death of these infants, but that this result is always brought about by suffocation.

We cannot reproduce here the evidence for and against these opinions, and must content ourselves with adding that a strong effort has been made, for example, by Poppel,[3] to re-establish the earlier view, as to the serious significance of meningeal hemorrhage in these cases. For ourselves we must confess that the arguments of Schwartz do not seem sufficient to overthrow entirely the old doctrine respecting the cause of the "apoplectic death and simulated death (Scheintod) of new-born infants."

The influence of the extravasation makes itself felt, under these circumstances, in various ways: the children are either born dead, or at first in a state of asphyxia, and die soon afterwards; or they recover somewhat, and live for a short time; or, finally, in rare cases, they recover. The symptoms of asphyxia, with which the children die immediately, or very soon after delivery, are too familiar to require further description. In other cases it is possible, by persistent efforts, to revive the respiration; but the infants remain very weak and somnolent, often even comatose, and die, after a few days, in convulsions. Sometimes these children seem very feeble and somnolent immediately after birth, and continue in the same state for from one to three weeks, after which coma and convulsions come on and close the scene. In cases of this kind, as in another class shortly to be mentioned, the assumption appears inevitable that the hemorrhage only gradually reaches a fatal amount. It happens, namely—though, to be sure, but seldom—that infants with this affection appear entirely well and lively immediately after delivery, but after the lapse of a few days fall suddenly into an apoplectic state, and die generally in convulsions.

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[1] *Schwartz*, Die vorzeitigen Athembewegungen. Leipzig, 1858, S. 293, ff.

[2] *Pernice*, Greifswalder med. Beiträge. 1863.

[3] *Poppel*, Ueber den Scheintod Neugeborener. Monatsschr. f. Geburtskunde und Frauenkrankheiten. 25. Bd.

Meningeal hemorrhages of any considerable amount probably always prove fatal; but if slight and circumscribed, they may be reabsorbed (Bednar, Virchow, and others). Whether or not large extravasations, capable of producing marked symptoms, can also be absorbed, is difficult of establishment, because the symptoms associated therewith are so little regular and defined, that the recovery leaves us in doubt as to the correctness of the diagnosis.

Prognosis.

It will have become evident, through the foregoing remarks, that the prognosis, whether in the case of adults or of infants, is very unfavorable. The accidental discovery of certain appearances at post-mortem examinations, in a few cases, proves that reabsorption of the extravasation and recovery, where the hemorrhage was very small, is a possible occurrence. For the cases in which the affection can be diagnosticated, however, death seems to be the inevitable termination. The establishment of the diagnosis must rest chiefly upon the recognition of the presence of favoring etiological conditions.

Treatment.

With adults the same rules of treatment are to be followed as were laid down for intra-cerebral hemorrhage (vide the appropriate chapter), but unfortunately, as has been remarked, with but little prospect of favorable results.

With infants, immediately after birth, the usual treatment for "simulated death" is to be employed. If they show signs of recovery, the principal aim should be to arouse the action of the heart, in order to secure a proper supply of blood to the medulla oblongata, and thereby to maintain the irritability of the vitally important centres situated there.

For this purpose the only thing to do is to provide nourishing food and a few drops of wine. No means are known which can directly further the reabsorption of the extravasation.

OCCLUSION OF THE CEREBRAL VESSELS.

L. Rostan, Recherches sur le ramollissement du cerveau. Paris, 1823.—*Bright*, Guy's Hosp. Reports. I.—*Ph. Fr. W. Voigt*, Ueber die Erweichung des Gehirns und des Rückenmarks. Heidelberg u. Leipzig, 1840.—*Durand-Fardel*, Traité du ramollissement du cerveau. Paris, 1843.—*Hasse*, Henle und Pfeuffer's Zeitschrift. 1846.—*Virchow*, in Traube's Beiträge zur experimentellen Pathologie und Physiologie. 1846. II. Bd.—*Puchelt*, Die Krankheiten des Venensystems, in Canstatt's Jahresbericht. 1845.—*B. Cohn*, Klinik der embolischen Gefässkrankheiten. Berlin, 1860.—*Traube*, Deutsche Klinik. 1854.—*Panum*, Ueber den Tod durch Embolie. Zeitschr. f. klin. Med. von Guensburg. 1856. 7. Bd.—*Lebert*, Virchow's Arch. 9. Bd.—*Gerhard*, Deutsche Klinik. 1857.—*von Dusch*, Ueber Thrombose der Hirnsinus.—*Griesinger*, Zur Lehre von den Hirnkrankheiten aus Otitis interna. Arch. d. Heilkunde. Jahrg. 3. XXIV.—*Heusinger*, Virchow's Arch. 11. Bd.—*Ruehle*, Virchow's Arch. 5. Bd.—*Lancereaux*, De la thrombose et de l'embolie cérébrales. Thèse. Paris, 1862.—*Prevost et Cotard*, Recherches physiol. et pathol. sur le ramollissement cérébral. Gaz. Méd. de Paris. 1866.—*Heubner*, Archiv der Heilkunde. 1870.—*Huguénin*, Pathologische Beiträge. Zürich, 1869.—*Bertin*, Etude critique de l'embolie dans les vaisseaux veineux et arteriaux. Paris, 1869.—*Feltz*, Traité clinique et expérimental des embolies capillaires. 1870.—*Russell Reynolds* and *Charlton Bastian*, Art. Softening of the brain, in Reynolds's System of Med. p. 446, et seq.—*Eliza Walker*, Ueber die Verstopfung der Hirnarterien. Inaug.-Diss. Zürich, 1872.—*J. Cohnheim*, Untersuchungen über die embolischen Processe. Berlin, 1872.—See also the bibliographical lists printed at the head of each of the preceding chapters.

The arteries, veins, and capillaries of the brain are liable to become occluded, and the occlusion in either of these sets of vessels may give rise to pathological conditions of various kinds. This occlusion is met with in the arterial system much oftener than in the others. The occluding mass may either be produced on the very spot (autochthonous thrombosis), or may be carried there from other parts of the vascular system (embolisms). We shall treat first of the

A.—Occlusion of the Cerebral Arteries.

The changes in the cerebral tissues, which result from the occlusion of arteries, are identical in great measure with those which were formerly described under the head of "Cerebral Softening," considered as an independent affection. The controversy as to whether and how far the changes which follow arterial obstruction and those comprehended under the name of "softening" correspond with one another, may be regarded as finally settled, at least up to a certain point. We cannot discuss here, in all its details, the entire subject of encephalomalacia, which has kept pathologists busy ever since the time of Lallemand's and Rostan's observations (1820), and which was only definitely settled through Virchow's masterly investigations into the subject of thrombosis and embolism. One point alone may be briefly referred to. Certain observers have firmly maintained the opinion that the so-called encephalomalacia is always an inflammatory process; especially Lallemand, Bouillaud, and, above all, Durand-Fardel, who (with a disregard for German literature which is characteristic of him), even after the date of Virchow's investigations, continued to recognize only an inflammatory softening. On the contrary, other investigators have pointed out how often arterial diseases occur in connection with this process, and have recognized, besides the inflammatory, a non-inflammatory form of the affection, due simply to disturbances of nutrition, the nature of which has been variously conceived of; among these are Rostan, Cruveilhier, Abercrombie, Carswell, Bright, Hasse, and others. The mode and conditions of origin of this latter form may now be regarded as thoroughly understood, especially since the time of Virchow's observations. No points involving any new principle have been developed since then, although a series of certainly very interesting investigations, in the direction already marked out, have been communicated, which will be referred to in the proper place. It is only very recently that, through the labors of Cohnheim, further important advance has been made towards a better understanding of the processes in question.

The changes due to the occlusion of arteries characterize then a large if not the largest part of the cases of red, yellow, or white encephalomalacia, properly so called. Besides this form, we have already pointed out, in treating of cerebral hemorrhage, that softening may take place secondarily in the tissues around the seat of an extravasation. Still other forms will be treated of in due season.

Etiology.

The source of the emboli which are carried into the cerebral arteries is to be sought in that segment of the vascular system which embraces the pulmonary veins, on the one hand, and the left side of the heart, the first part of the aorta, the carotids, and the vertebral arteries, on the other.

The emboli themselves are made up, as a rule, of blood clots, masses of fibrine, connective-tissue growths, or chalky concretions, swept from the place of their formation into the circulation. It does not lie, of course, within our province to discuss here the pathology of thrombosis and embolism in general. We need only speak of those conditions which result in the formation of these occluding masses, whether they are formed on the site of the occlusion or are carried thither by the blood.

The most important source of such emboli as concern us lies in *endocarditis*. This affection runs sometimes an acute course, in which case it is associated with ulceration; sometimes a chronic course, leading to the formation of warty, or villous excrescences, or to valvular insufficiency and stenosis: and either of these two varieties of the disease may, therefore, furnish an opportunity for the formation of emboli. We possess no accurate statistics which can determine which of these varieties most frequently leads to cerebral embolism, and can only make the following general statements with regard to the matter. Acute ulcerative endocarditis causes capillary embolism oftener than the obliteration of the larger arteries, though the latter now and then occurs. The carrying off of real connective-tissue formations into the circulation is an event of relatively infrequent occurrence. The common chronic endocarditis is the form most important in this connection, especially in the cases where, in addition to changes in the endocardium of ancient date, a fresh exacerbation of the inflammatory process has taken place.

An inflammation of the lining membrane of the left side of the heart, no matter where its seat, may give rise to the formation of emboli; nevertheless this result is much more common in connection with diseases of the valves than with those of the membrane lining the cavities; and, further, the atrio-ventricular orifice with its valves is much oftener affected than the aortic orifice.

Thus, for example, in the cases of Bertin the emboli originated four times in the left auricle, twelve times in the ventricle, ten times at the aortic valves, twenty-four times on the mitral valves; in nine other cases mention is made only of the "valves of the left side of the heart," without more exact designation. Affections of the heart may, however, lead to embolism in other ways, though much less frequently than through the agency of endocarditis. In certain rare instances this result follows myocarditis with its various sequelæ; still more rare are the cases such as one, for example, reported by Oppolzer,[1] where a syphilitic gumma in the cardiac substance, broke through into the sinus of Valsalva. It happens somewhat more frequently that particles crumble off from a *blood clot*, which has become developed within the heart, independently of any local inflammatory process, and are carried away as emboli. The most important instances of this kind occur in the cases where thrombi form in the left auricle in front of a contracted atrio-ventricular orifice. In the same category belong all those cases of cardiac thrombosis due to diminished efficiency of the heart's action, combined with retardation, or, still more, complete local stoppage of the circulation. The processes which produce these effects are either general diseases with marked cachectic diathesis (emboli originating in this way have been found in connection with carcinoma, tuberculosis, empyema), or diseases of the heart itself which greatly impair its power, such as fatty degeneration, myocarditis, the stage of incomplete compensation in valvular diseases. It is practically found that the clots form, as a rule, under these circumstances, in the auricle, and are carried thence into the peripheral arteries.

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[1] Wiener med. Wochenschrift. 1860.

In proportion to the frequency with which the formation of emboli is associated with affections of the heart, and especially endocarditis, the number of cases in which their origin is of a different kind is very inconsiderable. Aneurisms, especially of the aorta, form their next most common source. An interesting case is described by B. Cohn, in which an embolic mass in the cerebral portion of the carotid artery of six months' standing, furnished a second embolus, which crumbled off and was carried deeper into the brain. In very rare cases the thrombotic masses form within the lungs, and are swept thence by way of the left side of the heart into the brain; this may occur in cases of carcinoma pulmonum.

The *autochthonous thromboses* of the *cerebral* arteries may *originate* in either of several well-recognized ways. It has been proved experimentally that coagulation of the blood may be made to occur, or the tendency to its ocurrence increased, both through the presence of structural changes in the vascular walls, and through arrest of the circulation. These same two conditions are now met with as clinical phenomena. By far the greater number of cases of spontaneous thrombosis of cerebral arteries are seen in persons in whom diseases of the walls of the blood-vessels are also present. These diseases consist either in fatty degeneration, with destruction of substance, or in real inflammation of the coats of the arteries, leading, as it does, to sclerosis, ossification, or calcification. They are not always confined, in such cases, exclusively to the cerebral arteries, but occur also in other parts of the vascular system. Together with the affections of the vascular walls, sometimes also as a result of them, the second of the above-mentioned influences is apt to make itself felt — the slowing of the arterial blood-current. This may be to some extent a direct consequence of the vascular degeneration, since through this degeneration the elasticity of the vascular walls, which normally helps to maintain the rapidity of the circulation, is more or less impaired; and, further, since the calibre of the vessels is narrowed through the sclerotic changes which have taken place. The slowing of the blood-current through the cerebral arteries, necessary to the formation of thrombi, may, furthermore, be brought about by an enfeeblement of the heart's action. The atheromatous process, if seated in the coronary arteries, may play a part in the production of this result also, by interfering with the nutrition of the cardiac substance. We have referred to the other influences which tend to weaken the heart's action in speaking of the formation of clots within the heart. Again, autochthonous thrombi have occasionally been observed in the case of cachectic persons, where there was not the least appearance of alteration of the walls of the vessels, or any condition which might give rise to embolism. The coagulation is evidently to be referred, under these circumstances, to excessive diminution of the force of the heart's impulse.

Autochthonous arterial thrombosis, with consequent softening, is sometimes, though rarely, met with as a result of other causes. Thus Verneuil[1] relates a case where, in consequence of an injury to the neck, a rupture of the internal and middle coats of the internal carotid had taken place, attended with the formation of a thrombus, which spread even to the terminal branches of the middle cerebral artery. A similar state of things has been observed as a result of the compression of the carotid by tumors. *À priori*, we should expect to find a similar extensive occlusion taking place as a frequent result of ligature of the carotid. The infrequency with which this is found practically to occur is evidently to be explained by the fact that the circulation through the arteries forming the circle of Willis is so rapid as not to allow of the growth of the thrombus in them, by the deposition of new material; and, in the few cases in which softening has followed this operation, it is probable that the distribution of the collateral vessels (vide Ehrmann, l. c.) was somewhat anomalous. Thudichum and Bastian have maintained, that in leucocythæmia and kindred states the white globules of the blood may collect together into masses, and thus form thrombi or emboli, sometimes of considerable size; but this statement requires further confirmation before it can be adopted.

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[1] Bull. de l'Académie de Méd. 1872. No. 2.

Cases now and then occur in which the vascular occlusion is associated with extensive softening, where, for anatomical reasons, the conclusion seems inevitable that the thrombosis is of secondary nature (as the supporters of the inflammatory theory of encephalomalacia maintain to be invariably the case).

The predisposing causes of cerebral embolism and thrombosis may be summed up in a few words. First of all, the influence of advanced age is to be mentioned, which used to be considered as the most important cause of cerebral softening. We know now that this is only true of the softening from thrombosis, and that the influence in question is only an indirect one, due to the fact that in advanced years the various changes in the vascular system are met with, which both lead to thrombosis, and favor the destructive changes in the cerebral substance by interfering with the establishment of a sufficient collateral circulation. We do not mean to deny, at the same time, that autochthonous thrombosis occurs also in the earlier periods of life; in fact, we have ourselves observed several cases in persons between thirty and fifty. Embolism, on the other hand, is oftener met with in relatively youthful persons, though it may, of course, occur also in those of advanced years. Less important than the influence of age is that exerted by chronic alcoholism, although it is not improbable that it does predispose more or less to diseases of the arteries, and thereby to thrombosis and embolism. Still less certain is the influence of syphilis. Acute rheumatism, on the other hand, certainly plays an important part in bringing about the results in question, inasmuch as it frequently leads to endocarditis. The influence of sex is but trifling and indirect, consisting only in the fact that men are more liable to suffer from alcoholism and rheumatism. According to the figures obtained from certain collections of cases (those compiled, for example, by Gerhardt), women are more often affected than men; but the instances are not sufficiently numerous to make it clear that these results were not accidental. Finally, there is no clinical proof that there are any especial exciting causes which favor the setting free of emboli.

Pathological Anatomy.

The observations in this direction have shown that certain arteries are especially liable to become occluded, although this is true only of the occlusion by embolism, not of that by thrombosis. The liability to the latter affection is shared almost equally by a number of different vessels, especially the internal carotid, the middle and the posterior cerebral, and the vertebral. In the cases of thrombosis that have come to our notice it has happened that the last-named artery was oftener affected than the others; but this may have been purely accidental.

It is much more characteristic of embolism than of thrombosis, as already stated, that it attacks certain vessels with especial frequency. It has not yet become possible to prove this statistically, since the results obtained through investigations of this kind have still constantly to be corrected to include the newer observations that are being daily reported; we may, however, make the following general statement: Emboli are carried very much oftener into the left carotid than into the right, which is to be explained by the difference in the angle at which the left carotid and that at which the innominate is given off by the aorta. It happens only exceptionally that they become lodged below the circle of Willis, and in the majority of cases they are swept into the branch which forms the chief direct prolongation of the carotid, the arteria fossæ Sylvii. This artery has been found to be the commonest seat of these embolic masses. The right carotid is affected less often than the left, and the vertebrals still less often, as is easily to be explained on simple anatomical grounds.

It happens occasionally that the occlusion, whether of thrombotic or of embolic nature, involves a number of vessels at the same time. A number of cases have also been reported which show that embolism may be bilateral (for example, by Bristowe, Huss,[1] Gerhardt, and others), and that all the emboli may be carried to their destination either at one and the same time, or successively. It need hardly be said that, together with the cerebral arteries, those in other parts of the body may also be occluded at the same time.

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[1] *Huss*, nach Schmidt's Jahrb. B. XCV.

Through the recent investigations of Heubner (l. c.) and Duret (l. c.), we have learned to know more precisely than formerly the boundaries of the districts supplied by each of the various cerebral arteries, and understand better the laws of localization of the softening in the different cases. Heubner, to quote him briefly, has shown that the arterial system of the cerebrum may be divided into a basal portion (Basalbezirk), embracing the arteries of the ganglia of the caudex, with the neighboring parts of the "mid-brain" (Mittelhirn), and a "cortical portion" (Rindenbezirk), embracing the arteries of the entire cortex cerebri (excepting the gyrus uncinatus), together with the corresponding medullary substance.

This method of division is an important one, for the reason that the vessels of the "basal system" are "terminal arteries" (in Cohnheim's sense (vide below)), a point to which this observer has himself called attention; while, on the other hand, those of the cortical system anastomose freely with one another. As for the details of Heubner's results, we shall refer to those only which concern the distribution of the arteria fossæ Sylvii, so often the seat of emboli. This vessel supplies the entire nucleus lenticularis, a portion of the corpus striatum, the capsula externa, and the anterior branch of the capsula interna; and its terminal branches supply the second and third frontal convolutions, further, those portions of the central convolutions which are turned towards the convexity of the brain, parts of the three temporal convolutions, the parietal convolutions, and, finally, the island of Reil. It has been observed, further, that these same portions of the brain are the ones most prone to suffer from softening, which is greater or less in extent according as the main trunk of the Sylvian artery is occluded, or only one of its branches. Similar differences in the extent of the pathological change, dependent upon the varying seat of the embolus, are observed also in cases of softening in other arterial districts.

We may pass over the changes which go on in the embolus or thrombus itself, as they are in no wise different from those observed under similar conditions existing elsewhere in the body. One fact only need be mentioned, namely, that now and then, where the clinical symptoms point to the existence of an embolus, and where at the post-mortem examination softening is found, the affected vessel has been discovered to be entirely empty. It is probable that in these cases the occluding mass has been reabsorbed, a process which seems to require several months for its completion.

Let us now direct our attention to the pathological changes in the brain which the arterial occlusion induces. The first fact of importance in this connection is that, when the seat of the obstruction is on the cardiac side of the circle of Willis, anatomical changes in the cerebral substance very rarely result —never, in fact, unless the collateral circulation through the other vessels forming the circle is, for some reason, insufficient to counteract the effects of the circulatory disturbances. For further discussion on this subject, we would refer the reader to the chapter on Cerebral Anæmia, in which reference is made to the effects produced by ligature of the carotid. It is also conceivable that permanent anatomical changes would fail to occur in case the embolus should be carried onward into the arterial system of the cortex, where the circulatory disturbances are readily compensated for. When, however, the embolus is lodged in one of the terminal arteries of the basal arterial system, changes invariably follow, resulting either in simple necrobiosis or in hemorrhagic infarction. The immediate consequence of the occlusion of an artery beyond the circle of Willis is always anæmia, without softening of the parts which it supplies. This is followed either by hyperæmia, attended by œdematous swelling and hemorrhage (the presence of which formerly constituted the principal ground for regarding this entire condition as one of inflammation), the final result being a necrobiosis, softening of the affected district ; or the hyperæmic engorgement and the hemorrhages fail to occur, and the vascular obstruction is followed by simple necrobiosis, giving a whitish-yellow or white color to the affected mass.

The usual duration of these different stages is not precisely known. One fact alone is certain, namely, that the brain bears the circulatory disturbances resulting from vascular occlusion less well than other organs, and that structural changes occurring in it are more quickly induced. It may be said that, as a rule, these changes begin in the course of the second twenty-four hours after the occlusion, although isolated cases have been reported in which the consistence of the brain tissue was still apparently normal after the lapse of two days.

The first form under which, as stated, the structural changes occur has been designated as "*red softening.*" The volume of the affected portion of the brain seems at first to be increased, while its consistence is somewhat diminished. The color may be any one of the various shades of red between quite light and very dark. In the midst of the mass small points of a darker color are to be seen, which are the seats of "capillary apoplexies." These minute extravasations are at times so numerous and so closely approximated to one another as to give the whole mass the appearance of a hemorrhagic focus. As the changes progress, the consistence of the parts becomes gradually less, and the softening more and more evident. As a rule, the transition from the diseased to the healthy tissue is a gradual one, the line of demarcation never being so sharply drawn as in the cases of the ordinary form of hemorrhage, especially at the outset.

After the lapse of from two to four weeks the reddish color begins to fade away, and gives place to yellow (this stage represents one form of the "*yellow softening*"). This change of color arises from two causes: first, from changes which the coloring matter of the blood undergoes; and, further, in some degree in consequence of the fatty metamorphosis of the nerve elements.

If the patient lives long enough, this yellow softening may give place to a milky *white softening;* but this change takes place only after the lapse of several months. The affected tissue forms a mass which, as a whole, is semi-fluid, but contains a certain number of denser particles. As a final result, the constituents of this mass may be in part reabsorbed, leaving a sort of cyst filled with quite thin fluid. Small masses of disease may be completely reabsorbed. The construction of the larger cysts may be of the same character with that of those following hemorrhage; indeed, the two varieties are often not to be distinguished. For this reason, we refer the reader to the description already given of the cysts of the latter kind, in the chapter on Hemorrhage, where may be found also an account of the secondary changes which occur, in both sets of cases, in the brain and spinal cord.

Vascular occlusion is, however, not invariably followed by venous engorgement and infarction—in other words, "red softening." It may, on the other hand, be followed by simple necrobiosis, which presents from the outset the characteristic features of "*yellow softening.*" In this case also the consistency of the affected mass becomes lessened, its color yellowish; but this color is here not due to blood pigment, but to the presence of thickly crowded granular bodies, containing fat drops, which result from the retrograde metamorphosis of the various tissues (vide below). The further changes are of the kind already described.

Finally, cases are observed in which a condition of "*white softening*" exists from the very outset.

We will now consider the question, *under what conditions these various forms of softening arise*, especially the red softening, with its attendant engorgement and extravasation. The latter was formerly considered as the result of inflammation and collateral fluxion, until Virchow pointed out that the back-flow of venous blood into the district supplied by the obstructed artery was the important influence. We must pass over in silence the various theories proposed since then, in explanation of this process, preferring to pass at once to speak of Cohnheim's experimental investigations, which at present afford the best explanation of the different processes resulting from embolism.

Cohnheim has discovered, by direct microscopic observation, that when one terminal artery is occluded, the blood immediately begins to flow back from the nearest unaffected artery into the vein and the entire vascular system belonging to the district of the obstructed artery, in which the circulation has been brought to a stand-still; and that thence results the hyperæmic engorgement. Next follows the hemorrhage, consisting in a diapedesis of the red blood globules, which comes to pass in the following manner: when, in consequence of the back-flow of the blood-current through the veins into the other vessels of the affected district, the pressure there is made equal to that in the surrounding vessels, so that no further influx of blood, or renewal of the blood already collected, can take place, structural changes begin to occur in the walls of the vessels involved, and it is as a result of these changes that the escape of red corpuscles becomes possible. Such is the mode of development of the "red" softening.

Cohnheim has further shown under what conditions it may happen that the hemorrhage should fail to occur, and instead a state of "yellow" or "whitish-yellow" softening be present from the outset. The most important of these conditions consists in the coagulation of the blood contained in the vascular district of the obstructed artery, having the effect of preventing the in-flow of the venous current.

Cohnheim considers, further, that the position of the patient, by bringing to bear the influence of gravity, must play an important part in determining whether the venous blood, which at the best is under but slight pressure, shall flow into the district in question.

The force of the heart's action is also of material significance. We cannot go at greater length into the discussion of Cohnheim's investigations. They show that in every case the nature of the final result is determined by whether or not the establishment of a backward venous current is possible. If it is not possible, we shall have to do with a simple necrobiotic process within the district deprived of its blood-supply, unattended by hyperæmia and extravasation,—*i. e.*, from the very first, with a condition of yellow softening.

In general terms this entire pathological process is now universally regarded as of the nature of *necrobiosis*, and in no respect inflammatory. This view is further confirmed by the result of *microscopic examination.* Within the first twenty-four hours such examination reveals nothing abnormal (in the cases where engorgement and extravasation exist) except the presence of red blood-cells.

At a later period the nerve-elements undergo gradual degeneration, just as the fibres of a peripheral nerve do after section of the trunk. The next most prominent microscopic peculiarity consists in the presence of the so-called granular corpuscles, which Virchow believed to arise from transformation of the cells of the neuroglia.

We need not detail the various theories that have been proposed, respecting the genesis of these bodies, and of the recent monographs upon this subject; we would refer only to those of Ludwig Meyer,[1] who considered them to result from fatty degeneration of the cell-elements of the vessels, and of Huguénin,[2] according to whom the granular corpuscles originate: 1, from the nuclei of the neuroglia-cells; 2, from the cells composing the capillaries; 3, from the adventitia of the blood-vessels; 4, from the nuclei of the unstriped muscles of the blood-vessels; 5, from the connective-tissue nuclei of the perivascular lymph-spaces; 6, from the spindle-cells of the cortex cerebri; and, 7, in all probability from the ganglion-cells of the cortex. The retrograde metamorphosis of the affected tissues then gradually progresses, until, finally, the yellowish, semi-fluid mass contains only fatty detritus, drops of fat, and, in addition—supposing the process to have begun with red softening—blood-crystals.

Experimental Investigations.

The first experimental inquiries into the subject of thrombosis and embolism—or that part of it which concerns us—we owe to the labors of Virchow. Their results and their significance for the development of the entire doctrine of embolism are so familiar to all that we need only refer to them here in passing.

They have been followed by a number of more or less extensive investigations, some of them concerning the general subject of vascular occlusion, that of the cerebral vessels with the rest, and others entering more directly into the latter branch of the subject. It lies beyond the design of this treatise to enter into the general subject of embolism, and we will therefore only mention: that Panum studied the occlusion of cerebral vessels, especially with the view of determining the manner and cause of death; that B. Cohn investigated experimentally various clinical and anatomical points; while Feltz treated particularly of capillary embolism. Prévost and Cotard made a series of careful experiments bearing upon the subject of the obstruction of cerebral arteries and its relation to softening; and, finally, the important experimental and microscopic investigations of Cohnheim are to be mentioned, respecting the structural changes which follow arterial occlusion; so far as they concern us, we have spoken of these investigations above.

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[1] Arch. f. Psych. u. Nervenkr. III. Bd.

[2] Ibid.

It is not to be wondered at that the study of the effects of a mechanical occurrence, such as the occlusion of a vessel, which may be so easily reproduced in the laboratory, should, after having been once entered upon, continue to prove an attractive one to the experimental physiologists; or that, through the combined teachings of experimental and clinical investigation, we should have attained greater familiarity with this process than with most others.

Symptomatology.

If the symptoms which characterize the later stages of thrombosis and embolism, *i. e.*, the stages of softening, be compared together, they will be found to be identically the same; but with regard to the *first stage* this is not the case. On the contrary, the symptoms that characterize the onset of the two affections differ from one another in important respects, so that they may profitably be described apart.

Let us begin with *embolism.* In the first place, it is worthy of mention that the symptoms which attend the occlusion of a cerebral vessel by an embolus always make their appearance suddenly.

It is plain that from the very nature of the process no prodromal signs on the part of the brain could possibly occur. The case is one, as it were, of injury to the brain from an entirely external cause. If prodromal signs are apparently met with in any case, they must be due, not to embolism, but to other accidental causes.

Although the symptoms are always sudden in their occurrence, their character, however, is liable to more or less variation. As a rule, an *apoplectic condition* is immediately induced. The patient, who may the moment before have felt himself in perfect health, or at least have shown no indication whatever of cerebral disorder, is attacked, without apparent cause, by dizziness, or he utters an involuntary cry, or complains for a moment of headache, or, even without the occurrence of these symptoms, which at any rate are of but momentary duration, he loses his consciousness.

This apoplectic attack resembles precisely in character that which was described as resulting from hemorrhage; and although there are certain symptoms which belong more especially to one or the other affection, yet there is no feature which may not be present in either case; and we are, therefore, justified in saying that the two sets of symptoms are in the main identical—in other words, that we are not able to judge, from the occurrence of the apoplectic attack alone, to which cause it owes its origin. We refer, therefore, for its description to that already given in connection with cerebral hemorrhage.

The differences which, a few years ago more than now, were claimed to exist, for example by Cohn, between the apoplectic attacks of the different origins referred to, are essentially the following:

In embolic apoplexy the patients were said to turn pale in the face; in the hemorrhagic apoplexy, red. We have called attention, however, to the fact that, in the latter variety, the patient's face may appear like that of a person in a faint; and, conversely, it may be red in the former case.

Pulsation of the carotids and stertorous respiration may also accompany embolism; and the pupils have also been observed to become unequal. Great weight is is still sometimes attached to the fact that in embolism consciousness is lost but for a brief period. This is well known to be true also of some cases of hemorrhage, however; and, on the other hand, the coma from embolism sometimes endures for several hours, and is finally terminated by death. In a case of our own, the patient being to be sure epileptic, the loss of consciousness was of five days' duration. Nevertheless, as a rule, it is certainly true that the duration of the apoplexy from embolism is less than that of the apoplexy from hemorrhage.

Although by far the greater number of cases open suddenly with an apoplectic attack, yet exceptions to this rule sometimes occur, of which of course only the more important can find mention here.

A number of cases have been reported in which the loss of consciousness was preceded by paresis of the muscles on the side opposite to that of the lesion, especially those of the face, either alone or together with those of one arm or one leg, or a disturbance of the power of speech.

Further, quite a number of cases could be cited, where the *coma was either entirely wanting*, or was replaced by dizziness, and slight confusion of mind of only a few seconds' duration; and the same point that was brought forward in connection with hemorrhage, may be repeated here, namely, that the localization of the lesion makes no difference for the result in this respect; at any rate, we know that embolism of the Sylvian artery, although as a rule attended with symptoms of apoplexy, may yet take place without them.

It is not uncommon for embolism of the cerebral arteries to give rise to *convulsions*. This fact has been established by various observers (among them Bristowe, Lancéreaux, Gerhardt, Prévost and Cotard, Biermer), so much so that Eliza Walker records, in her dissertation upon this subject, that out of ninety-seven cases of occlusion of the cerebral arteries, convulsive phenomena of some kind occurred, either at the outset or at a later period, in twenty-four. In the majority of cases these bore the form of typical epileptic attacks; in others, of convulsive movements in the muscles of the side afterwards to become paralyzed.

When the convulsions were of this latter variety, they preceded, in some cases, the occurrence of the paralysis; whereas the general epileptic convulsions invariably came on synchronously with the loss of consciousness and the paralysis. We shall speak later of the symptoms of this class occurring at subsequent periods.

For the occurrence of the convulsions, also, the seat of the embolus has been observed to be of relatively slight importance; in the cases reported it was found sometimes in the carotid, sometimes in the artery of the fossa Sylvii, sometimes in the basilar artery. They are, to be sure, especially common in cases of bilateral embolism.

We have not yet exhausted the list of the symptoms attending the onset of the attack. Vomiting sometimes accompanies apoplexy from embolism, as it does that from hemorrhage; and one cause of this, in certain cases, seems to lie in the peculiar position of the basilar artery. Further, the occurrence of the embolism is sometimes marked by delirium, even of a very pronounced kind, accompanying the paralysis, and disappearing after a few hours. Cases of this kind are, however, exceptional; a few such have been reported by Hammond. Finally, cases have occasionally been met with, where no other diagnosis but that of embolism could be entertained, in which the patients were suddenly attacked—entirely without warning—by aphasia, unattended by other symptoms of any kind, which disappeared again in the course of one or two days.

We will next turn our attention to the question, what is the cause of the apoplectic attack in cases of embolism? It need hardly be said that the influences present in these cases are different from those present in cases of hemorrhage.

The answer that is usually given to the question is, that the loss of consciousness is a result of the anæmia. The affair does not seem to us, however, to admit of being dealt with so simply. In the first place, it might be asked how it happens, if this is the case, that ligature of the carotid—which certainly causes a much more extensive anæmia than occlusion of a smaller artery (for example, the Sylvian)—so rarely induces loss of consciousness.

The force of this objection is not diminished by the rejoinder, that this is to be explained by supposing a collateral circulation, through the circle of Willis, to become established, since the same might be said for the cases of occlusion of the vertebral artery; and yet in them apoplexy is of common occurrence. Furthermore, it is questionable whether it is possible that anæmia of so small a portion of the brain as is usually affected in embolism, even if suddenly brought about, could, as such, induce loss of consciousness. Certainly no proof has been given of the possibility of this occurrence; nevertheless it is true that the force of the criticism is somewhat weakened by a consideration of the results of Heubner and Duret's investigations, which have demonstrated the existence of abundant anastomoses between the arteries of the cortex cerebri, but have shown, at the same time, that marked and extensive disturbances of circulation and changes in pressure may occur in this region at the moment of the occlusion.

The collateral hyperæmia cannot be the cause of the apoplexy, for it also is evidently too insignificant. In case of bilateral embolism the sudden anæmia may, in virtue of the size of area which is involved, be the principal factor in bringing about the result in question; but for the majority of cases some other influence must, in our opinion, be regarded as the essential one.

Attention was called, in the course of the discussion upon hemorrhagic apoplexy, to the doctrine of the so-called "étonnement cérébrale," upheld by Trousseau, Jaccoud, and others, as being one which commended itself to notice.

It may be that a similar influence helps to produce the apoplectic symptoms in cases of embolism, acting possibly through the agency of the sudden reduction of pressure in the affected district of the brain. We are entirely ready to acknowledge that this explanation is unsatisfactory; but, for the time being, we have no better one to offer.

The same difficulty attends the effort to explain the occurrence of the convulsions in these cases. Where the arteries of both sides of the brain are obliterated at once, or the basilar artery is affected, it would seem reasonable to regard the anæmia—in the one case because it is very extensive, in the other because it involves the pons Varolii and the medulla oblongata—as their cause. This cannot apply, however, to the cases where small arteries, other than this—for example, the middle cerebral—are the seat of the embolus. The convulsions occurring in these cases appear explicable only through the aid of the recent experimental investigations, proving the irritability of the cortex cerebri, according to which either the electrical (Fritsch and Hitzig, Ferrier, Dupuy) or the mechanical (myself) excitation of certain definite regions may call out more or less widespread convulsive movements. Reasoning from analogy, we might assume that the sudden anæmia acted as an irritant to those same regions of the cortex, and with the result indicated. We have not space here to discuss the subject more at length.

The clinical phenomena which attend the *occurrence of thrombosis* are usually, though not invariably, of a different character from those seen in the same stage of embolism. The occlusion of an artery by autochthonous thrombi is preceded, as a rule, by premonitory symptoms of various kinds. These symptoms have their origin in the gradual reduction which takes place in the calibre of the affected vessel, and can only be called premonitory from the point of view of their relation to those which accompany the final complete occlusion, being in reality symptoms of incomplete occlusion. It would be therefore more strictly correct to say that in thrombosis (in contradistinction to embolism), the symptoms are of slow and gradual development.

These so-called premonitory signs may be of various descriptions. The commonest are pains in the head, which are generally diffused, though Hammond states that they are often referred to the neighborhood of the morbid process ; and next, in order of frequency, dizziness and a sense of general confusion. Further, mental disturbances are met with, among which failure of the memory is especially prominent. The patient complains also, not unfrequently, of abnormal sensations, of the nature to numbness, coldness, formication, or "pins and needles," but rarely of actual pain.

These sensations are usually felt only in one extremity, or throughout the distribution of one nerve, but they may involve an entire half of the body. Motor disturbances are observed : these are usually of the nature of more or less extensive paresis, but now and then the loss of motor power is preceded by slight convulsive movements. The differences in the extension of the symptoms are plainly determined by the size and physiological importance of the occluded vessel.

The development of these paralytic symptoms up to their maximal intensity may be either very gradual, or the paralysis may come on all at once ; or, finally, the paralytic symptoms may increase by sudden steps, remaining uniform for a certain period between each two.

The final attack may be attended, as in the case of embolism, by apoplectic symptoms, or it may occur without inducing any loss of consciousness whatever. The duration of the prodromal stage is likewise variable : we have seen cases of autochthonous thrombosis (the diagnosis having been afterwards confirmed by the autopsy) where only twelve hours, and others where several months, elapsed between the time of occurrence of the first cerebral symptom and that of the apoplectic attack. Now and then it happens that thrombosis, like embolism, comes on suddenly, entirely without warning; but such cases are very exceptional.

The *further history* of these cases of occlusion from thrombosis or embolism, of which we have now described the mode of onset, may vary greatly in character: the symptoms may terminate either in death or in complete and permanent recovery; or, after more or less improvement, the attack may recur; or, finally, the district supplied by the occluded vessel may undergo softening, attended with loss of its physiological functions and the production of permanent morbid symptoms. We will speak first of the last-mentioned occurrence.

According to the observations of Bourneville (l. c.), the temperature, under these circumstances, begins to rise on the second or third day, and may reach the height of 40° C. (104° F.); after two or three days, however, it begins again to fall, sinks rapidly, and finally becomes stationary. Except for the rapid decline of the curve, this behavior of the temperature is the same with that seen in the so-called period of reaction in cases of hemorrhage.

It is interesting to compare these observations with those of the earlier observers, especially Cohn. It was formerly believed that cases of cerebral softening could be distinguished from those of hemorrhage by the presence of a *diminished temperature of the body at large*, which, it was said, could be detected not only at the outset of the case, but at later periods as well. The investigations of Bourneville have, however, shown the incorrectness not only of the latter's statement, but also of the former's, the supposed initial fall of temperature in encephalomalacia being either entirely absent, or, if present, less pronounced than in cases of hemorrhage.

Supposing the stage of necrobiosis to have become thoroughly established, the symptoms with which we have to deal will be those of the localized cerebral diseases in general. Much labor has been spent in looking for differences between the symptoms of softening and those of the chronic stage of hemorrhage; but it is now pretty well agreed that essential points of difference do not exist between them. All the symptoms that characterize either process may be met with in the other as well; the differences between the special cases arise from differences in the position of the lesion; and, the fact, that certain symptoms are of specially frequent occurrence in one or the other of the two affections, is to be accounted for in a similar manner, namely, by the fact that the hemorrhage and the softening have each their favorite seats. We would emphasize, therefore, again the fact that the clinical results of *softening and of hemorrhage, regarded as localized diseases, may be identical.*

To avoid repetition, we refer the reader, therefore, to the chapter on Hemorrhage, pausing here only to call attention to a few important points.

The *motor-paralysis* from thrombosis and embolism, is generally of the same character with that seen in affections of the nucleus lenticularis, corpus striatum, and neighboring parts, *i. e.*, the muscles of the opposite extremities and opposite side of the face are involved. Those of the right side are oftener affected than those of the left, which arises from the greater frequency with which emboli become lodged in the left than in the right Sylvian artery.

If other vessels are occluded, the paralysis will involve other nerves than those mentioned. All the different cranial nerves have, at one time or another, been found involved, and in very exceptional instances, motor-paralysis of every kind has been wanting.

It will be evident, from a simple consideration of the nature of the process in question, that isolated destruction of the tracts corresponding to a single nerve will occur less often here than in the case of hemorrhage. Bilateral paralysis of the extremities, of the muscles of the neck, and of the facial muscles, has been observed as a consequence of bilateral vascular occlusion (Gerhardt).

In former times great significance was attached to the absence of contractures in the paralyzed limbs, in this affection; Lancéreaux, for example, speaks of them as being so rare as scarcely to deserve mention. Durand-Fardel, to be sure, pointed out that this view was an erroneous one; but the force of his statements is weakened by the fact that he includes the cases of necrobiotic and of encephalitic softening in the same category.

It is, however, true that contractures, if not so common as in cases of hemorrhage, still occur often enough, and that their absence in any particular case cannot be regarded as of pathognomonic significance. We have ourselves observed cases of this kind, and they have been frequently reported by others. In all other respects the stationary embolic paralyses are precisely like those due to hemorrhage, one point of difference alone calling for notice, which is, that a few cases have been recorded, and our own experience can furnish a very striking one of the kind, in which, for a long time after the occurrence of the paralysis, convulsive movements were observed to take place in the paralyzed muscles, occasionally associated with a well-marked epileptic attack. We are unable to offer a satisfactory explanation of this singular occurrence, which, to the best of our knowledge, has not been met with in cases of hemorrhage.

As for the disturbances of the sensitive, the trophic, and the vaso-motor functions, it is all-sufficient to refer the reader to the description given of them as they occur in connection with cerebral hemorrhage.

The same is true of the affections of the *special senses*, except that the two sets of cases differ somewhat in respect to the disturbances of *vision*.

In a very few cases of embolism, in connection with the other symptoms, amaurosis, from blocking up of the ophthalmic artery, has been observed to occur with great suddenness. On ophthalmoscopic examination the signs of anæmia of the fundus oculi have been found, with narrowing of the arteries and veins. Even in the cases where no diminution of the normal power of vision has been present, however, ophthalmoscopic examination often furnishes a help to the establishment of a diagnosis, to which certain observers—for example, Hammond—attach much weight. When, for instance, as so often happens, the middle cerebral artery is occluded, a collateral fluxion in the distribution of the ophthalmic artery may result, causing arterial and venous hyperæmia of the retinal vessels and congestion of the optic disc.

The disturbances of the *intellectual faculties*, consequent upon embolic softening, may be of various kinds, as was stated to be true of those following hemorrhage.

This would be the proper place to speak of a symptom which is so often associated with cerebral embolism that it is generally discussed in connection with it—namely, *aphasia.* Since, however, this subject will be found treated of at length in another part of this work, we may omit to deal with it here.

It need only be mentioned that the reason for the so frequent occurrence of aphasia in these cases lies in the fact that the embolism of the left Sylvian artery, which is the commonest form, induces functional disturbances in that district of the cortex cerebri, with the affections of which the disorders of speech are regularly associated.

For further details, with regard to the matter, we must refer to the section treating of Aphasia. It is an error to suppose, as has already been pointed out, that aphasia occurs only in connection with embolism or thrombosis; for it also accompanies hemorrhage, of which, indeed, it may even be the only symptom which is present.

All the symptoms which we have described may persist permanently; in fact, after they have lasted for more than a few weeks, complete recovery from them is not to be looked for, though a certain degree of improvement, in respect to the paralysis, may still be possible. In these respects, therefore, the condition of the patients is precisely like that of those suffering from the result of hemorrhage.

The clinical history of cases of thrombosis and embolism, is, however, not always that which has been described. In some instances, namely, *an improvement takes place in the paralytic symptoms*, although they may have been just like those accompanying pronounced softening. The possibility of the occurrence of this result plainly turns upon whether the cerebral tissues, robbed of their nutriment, can be sufficiently rapidly supplied with the needful amount of arterial blood by way of the collateral circulation. If this is the case, a rapid improvement may occur, noticeable after the lapse of even a few days.

Where this improvement concerns the paralysis, not only of single nerves, but of all the affected parts, its occurrence may even help us to distinguish the disorder under consideration from hemorrhage; for in the case of the latter, although it may happen that single nerves, whose central fibres were not actually severed, should resume their functions after a few days (vide the appropriate chapter), it is impossible that the complete recovery of all the parts involved should take place in so short a period. After once recovering, the patients may remain permanently in good health.

It is evident that in the cases where rapid improvement takes place, anatomical changes cannot have proceeded to the point of softening; and the primary loss of function of the affected part must, therefore, have been a result of the anæmia.

In a third series of cases, the patient, partially or completely recovering, is attacked anew with embolism, which may result either in death or in permanent paralysis; or in recovery for a second time, to be followed again in its turn, by a third attack. The duration of the intervals between the attacks may vary from weeks to years. In still other cases the symptoms may be at first of moderate severity, and afterwards, by sudden accessions, become more and more severe: this occurs oftener with thrombosis than with embolism.

Finally, death may follow immediately on the attack. It is noticeable, however, that this result does not take place after so short an interval as in some cases of hemorrhage. Only a few cases are on record which have ended fatally within the first twelve hours; and, as a rule, the patient keeps along without much change, remaining comatose, either persistently, or with a short interval of semi-consciousness, for a number of days, until finally he passes away, either in consequence of intercurrent pneumonia or without any such special cause.

Concomitant symptoms.—These arise from affections of the circulatory apparatus, or from embolisms occurring in other vessels at the same time with those in the brain.

For an account of the former group, we would refer to the chapter upon the etiology of Thrombosis and Embolism. It will be gathered, from what was there stated, that we may have to do with symptoms such as accompany valvular diseases of the mitral or aortic orifice of the heart, or endocarditis ulcerosa, or with the indefinite signs of inflammatory or syphilitic affections of the muscular substance of the heart, or with the symptoms of aortic aneurism.

In other cases we find the evidences of great enfeeblement of the heart's action due to some one of the above-mentioned general cachectic diseases. In cases of autochthonous thrombosis, it is often possible to discover at the same time an affection of the radial and other arteries, though not always, for the cerebral vessels are often attacked alone or with disproportionate severity. Sometimes no concomitant symptoms are present, or, to speak strictly, are discoverable.

The symptoms of embolism, occurring at the same time in different organs, are, when present, even more striking than those already mentioned. Most important among them are those of embolism in the spleen, the kidneys, and the arteries of the extremities.

This is, however, a subject into which we cannot of course enter, even briefly, in this connection.

Differential Diagnosis.

Traube[1] was the first to set down with accuracy the signs which justify the diagnosis of cerebral embolism. Since then many efforts have been made, from various sides, to establish definite points of difference between thrombosis and embolism on the one hand, and between these affections and hemorrhage on the other. Just as often, however, the ground has been taken, on which we also have placed ourselves, namely, that the diagnosis of hemorrhage, or of embolism or thrombosis, cannot in any case be unreservedly made. Under certain conditions it may be established, to be sure, with a probability which borders on certainty, and indeed with greater positiveness—supposing all of the characteristic symptoms to be present—in cases of softening than in those of hemorrhage. In other cases the probabilities on the two sides may be equal, and the diagnosis can rest only on conjecture.

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[1] Ges. Beiträge zur Pathol. u. Physiol. II. Bd. S. 281.

It has been made sufficiently clear by the foregoing statements that the cerebral symptoms by themselves are quite insufficient to enable us to draw the distinction in question, since there are none of them which may not occur in either of the affections under consideration, and we need not therefore go over the same ground again. Since, as has been shown to be the fact, hemorrhage, as well as embolism, may occur suddenly, without warning, and since aphasia may be caused by the former affection, and is not always present with the latter—since, further, embolism does not always cause paralysis of the right side of the body, but at times of the left instead—we do not see why any serious diagnostic importance should be attached to these points. Again, since the duration of the stage of coma in either case may be long or short, and since contractures of the muscles of the paralyzed limbs may come on, at a later stage, in either case, the fact that these symptoms are more common in the one affection than in the other should not be considered as constituting a reason for justifying a definite diagnosis in a special case. One circumstance alone seems to be of almost, though not absolutely, decisive importance: when, namely, a condition of well-marked hemiplegia, occurring coincidently with the attack, almost or quite disappears within a few days after it, the existence of hemorrhage as a cause would be very improbable.

It will have become sufficiently clear also, from what was said in the chapter on Symptomatology, that the presence of paleness or redness of the face, of pulsation of the carotids, of vomiting, and of the other symptoms of cerebral pressure, affords no certain means of diagnosis.

The case is different with regard to the so-called concomitant symptoms. If it is possible to demonstrate through them the existence of pathological conditions which might give rise to embolism, presumptive evidence is furnished for assuming that this is the process with which we have to deal.

Thus—to take the most striking case—if valvular diseases, or aneurism, were present, the diagnosis of vascular occlusion would receive a certain probability in its favor; whereas the presence of the other conditions, mentioned above as sometimes resulting in embolism, would be of less significance. At the same time the fact need not be dwelt upon that patients with heart disease may be attacked, as well as others, by cerebral hemorrhage. If, further, besides the valvular diseases, symptoms were present which made it probable or certain that embolism had taken place in the arteries of the kidneys, spleen, or limbs, there would be but little danger of error in referring an attack of apoplexy, if such should occur, to a similar cause.

The age of the patient is also to be considered, although it is not a certain guide, since both affections may occur either in youthful or in aged persons; hemorrhage, however, is less often met with in the case of the former.

Through these means it is sometimes possible, in especially favorable cases, to distinguish between embolism and hemorrhage; but we regard it as almost impossible to distinguish between hemorrhage and autochthonous thrombosis in a given case. The labor expended in this direction has been entirely fruitless, since all the means of diagnosis which have been proposed have proved unsatisfactory; and if the diagnosis of hemorrhage turns out to be in most cases correct, as is the fact, the reason is a very simple one, viz., that hemorrhage is a much more common occurrence than autochthonous thrombosis. Such being the case, we need not discuss the details of the matter further.

Prognosis.

After what has been said, it is plain that embolism and thrombosis of the cerebral arteries are always of serious import. Although it is true that the subjects of embolism may be youthful persons, in full vigor, yet these favorable circumstances will be overbalanced by the importance of the underlying affection to which the embolism was due, and the probability that similar attacks will recur in the future. In the case of autochthonous thrombosis, besides the danger that the occlusion will become more and more extensive, there is also that associated with advanced age and enfeeblement of the heart's action. At the outset of, and during, the primary attack, no prognosis can be given as to the probable course of the case, except that its severity is likely to be proportionate to the extension and severity of the primary symptoms. If the paralytic symptoms disappear after a brief period, there will be no reason to fear the presence of serious structural disease, but the chance of future attacks cannot be excluded. If the disease has progressed to the stage of softening, the prognosis will be the same with that already given for hemorrhage in its chronic stages, and must be influenced, evidently, by various conditions, among which are the age and constitution of the patient, the size and position of the lesion.

Treatment.

Prophylactic treatment is out of the question; we cannot even hope to remove the patient from the influence of exciting causes, because, as has already been said, we have no certain knowledge of the nature of such causes. It is only when premonitory symptoms are present for a long time, in connection with a slowly forming thrombus, that prophylactic measures become of service. These should of course be of the opposite kind to those applicable in case of threatening hemorrhage. The indication evidently is to further the establishment of an efficient collateral circulation by stimulating the heart's action. How can it be decided with certainty, however, whether the premonitory symptoms, in a given case, point to thrombosis or to hemorrhage? Since this is simply impossible,—since, further, hemorrhage is of commoner occurrence than thrombosis, and since an unseasonable stimulant treatment is likely to do more harm in a case of approaching hemorrhage than *moderate* derivative treatment, in the case of thrombosis, the practice most to be recommended would be to act as if the diagnosis of hemorrhage were certain.

The same diagnostic doubts increase the difficulty of determining the proper treatment for *the symptoms which characterize the primary attack.* Traube was the first to lay it down as a general rule that in the cases where the diagnosis can be determined with certainty, the treatment should be of a tonic and stimulant character, tending to hasten the establishment of the collateral circulation. During the stage of coma the same means should be used as during the analogous stage of hemorrhagic apoplexy. Subsequently a moderately stimulant treatment (wine, coffee, soup, nourishing food) should be used, regard always being had at the same time to the necessities of the particular case. Thus, for example, in the presence of hypertrophy of the left ventricle, or in the case of embolism occurring in robust persons, it would be necessary to be cautious in the use of these agents. If the temperature should become elevated, or if headache and other symptoms should occur, pointing to a severe collateral hyperæmia, local bleeding, derivation by way of the bowels, or applications of ice to the head might be called for.

Supposing no improvement to take place,—on the contrary, softening to occur, followed by the various symptoms which accompany circumscribed cerebral disease,—the same method of treatment would be applicable which was described in treating of the chronic stages of hemorrhage, and the statements there made need not be repeated.

B.—Thrombosis of the Cerebral Sinuses.

Isolated observations with regard to occlusion of the cerebral sinuses are to be found in the earlier medical literature; but the credit of having formulated it as a special disease belongs to Tonnelé. Next in order come a series of clinical observations. The investigations of Puchelt did not receive the notice which they deserved; those of Lebert on inflammation of the cerebral sinuses attracted more attention; and the more recent treatises of von Dusch, B. Cohn, and Lancereaux assured to thrombosis of the sinuses a permanent place in our nosology, as a separate pathological entity. At the same time and at later periods our knowledge of the disease, especially in point of symptomatology, was increased through the labors of various observers; we would refer particularly to those of Gerhardt, Griesinger, Corazza, Heubner, and Huguénin.

Etiology.

We must refrain from discussing in this connection, as in the case of arterial thrombosis, the conditions which lead to the formation of thrombi in general, and shall speak only of those causes which specially concern that part of the subject with which we have to deal. The thromboses of the cerebral sinuses may be divided, from the point of view of etiology, into two groups—those where the walls of the veins remain intact, and those where the thrombosis originates in phlebitis.

The cases of the former group usually originate in conditions of the nature of *marasmus* ("marantische Sinusthrombose"), in which the action of the heart is enfeebled, and the vis-a-tergo for the venous circulation materially weakened. This weakening of the circulation is probably of greater consequence in inducing the coagulation of the blood than the "thickening of the blood" itself, which is also spoken of as often exerting an auxiliary influence in this direction. The well-known anatomical peculiarities of construction and relations of the venous sinuses, the fact that they are rigid and incapable of collapsing, are traversed by bands of connective tissue, and have no muscular walls to further the flow of blood, explain why it is that the coagulation should take place in them with especial readiness when the heart's action is weakened. The marantic thromboses are particularly common among children, as Gerhardt, among others, has pointed out, especially during their first half-year of life, when they are prone to suffer from sudden collapse induced by severe diarrhœas. They may also be brought about in adults through the influence of any of the various conditions which materially enfeeble the action of the heart, such as profuse suppuration, cancer, marasmus senilis, etc. This form of thrombosis is especially common in the sinus longitudinalis and the sinus transversi.

It is difficult to decide whether *venous stasis*, due to impeded return of the venous blood towards the heart, can bring about coagulation in the cerebral sinuses. Cases certainly occur now and then which seem to strengthen an assumption of this kind. One such is reported by B. Cohn (l. c. S. 167); another by Langenbeck (Journ. f. Kinderkrankheiten, 1861). When we reflect, however, how often severe and protracted disturbances of respiration or repeated epileptic attacks occur, and how rarely they give rise to thrombosis of the sinuses, it can hardly be doubted that the effect in question can be brought about in this way only in the presence of certain other special conditions. In the two cases mentioned, for example, the patients were both enfeebled persons.

In the cases of the second group the thrombosis is due to an actual *phlebitis sinuum.* Proof is wanting for the fact that this condition can arise as a spontaneous and primary affection —or, to say the least, we are obliged to call in question the cogency of most of the observations hitherto made, on the ground that the cases are of too complicated a nature.[1] In almost every case some special cause for the phlebitis may be detected. The most common among them is disease of the cranial bones, especially the caries of the petrous portion of the temporal bone which accompanies otitis media, no matter of what origin. The cerebral diseases which arise from caries of the temporal bone are of various kinds, as is well known; but the most common among them—commoner than meningitis and cerebral abscess—is thrombosis of the sinuses.

It is plain that the sinuses which lie in the immediate neighborhood—the sinus transversi and petrosi—are likely to be first, if not exclusively, attacked; but, later, the process may involve the sinus cavernosus, sinus circularis Ridleyi, and even the upper part of the vena jugularis interna. In most cases of this kind a real phlebitis is induced, followed by the formation of purulent thrombi; yet it seems possible for simple thrombosis to take place without previous affection of the walls of the veins.

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[1] The most convincing case of the kind is one given in the Inaug. Diss. of A. Girard, Würzburg, 1864 (Fall von primärer Thrombose der Hirnsinus); but even this is not absolutely so, from the fact that "the cellular tissue about the gland. pituitaria and along the arcus, as far as the foramen magnum, was infiltrated with muco-pus, thickened and opaque.

Caries of the other cranial bones, of whatever origin, may also give rise to thrombosis of the sinuses. This may also follow *injuries*, especially if they cause inflammation of the diploë. In such a case the position of the lesion will of course determine which sinus is to be the seat of the thrombosis.

Inflammatory processes in the immediate neighborhood of the cranial bones may also give rise to phlebitis and subsequently to thrombosis of the sinuses. The most important among these are the much-dreaded large *furuncles* in the face, especially on the upper lip and forehead; further, *erysipelas of the head and face* may follow a similar malignant course.

B. Cohn observed a case in which suppurative phlebitis of the cavernous sinus occurred in connection with purulent inflammation of the deep muscles of the neck. Tonnelé has seen thrombosis from pustular eruptions on the head, a connection which is certainly not presumptively improbable. In all these cases, also, the thrombotic process in the sinuses may be entirely of a non-inflammatory character, arising, probably, per contiguitatem, as the secondary result of the affection of veins lying outside in the diseased tissues.

Pathological Anatomy.

Any sinus may, of course, become the seat of thrombosis, yet it will have become clear, from what has just been said, that certain of them are especially prone to suffer in this way: the sinus longitudinalis superior in the cases of marantic thrombosis; the sinuses in the neighborhood of the petrous bone in those of the phlebitic variety. The process may then remain confined to its place of origin, or may spread further per contiguitatem. The most common instance of this latter kind is the extension through the transverse sinus into the jugular vein. Inasmuch as thrombosis of the cerebral veins does not differ—as regards the structure of the thrombi themselves, the changes which go on in them, and their relations to the vascular walls—from that of the other veins of the body, we need not enter into these points here at length, nor need we discuss the possibility of the sinuses still remaining more or less permeable, and the various other contingencies, which are common to all cases of the kind in question.

Apart from the condition of the sinus primarily affected, certain other pathological phenomena may make their appearance, phenomena that are to be regarded essentially as the results of the venous stasis. These are especially prominent in cases where the superior longitudinal sinus is occluded.

The entering veins are found crowded with blood, enlarged, and often themselves filled with thrombotic masses, so that when seen lying on the surface of the brain they may resemble earthworms. It is not uncommon for rupture of the vessels to occur, causing hemorrhage into the meninges, which is at times very profuse, at times only such as to form spots of greater or less size on the surface of the brain. The amount of the fluid in the cavity of the arachnoid and in the ventricles may or may not be increased. These differences between the different cases evidently depend upon peculiarities in the seat of the thrombus, and the rapidity with which it forms, as well as upon the presence or absence of meningitis.

Besides the membranes, the cortical substance of the brain is liable to suffer injury, especially the convex portion of the hemispheres in cases of thrombosis of the superior longitudinal sinus. Capillary hemorrhages are of very common occurrence, and we have seen them in such numbers that the surface of the brain looked in parts as if peppered with them. Their favorite seat is the cortex. Lancereaux describes, as a further complication, small spots of softening, that, according to him, differ from the softening due to arterial obstruction in being of very small size, and in the greater frequency with which they occur in the gray matter and on the convexity of the hemispheres.

The phlebitic thrombosis is very often accompanied by meningitis, though there is no causal relation between the two processes, both of them being rather due to the same primary influences. Further, that meningeal hemorrhages and capillary apoplexies are so uncommon in cases of occlusion of phlebitic origin, arises from the fact that the seat of that affection is so generally such as we have stated.

Symptomatology.

It would be impossible to describe the symptoms of thrombosis of the sinuses in definite terms, for the reason that it is only very rarely possible to diagnosticate the presence of the affection, its own peculiar symptoms being usually masked by those of complicating disorders, or, where this is not the case, being indefinite and changeable in character. Apart from the influence of the position of the occlusion, its nature, *i. e.*, whether it is of phlebitic origin or not, is of chief significance in determining the clinical characteristics of any particular case. For the sake of greater clearness, it will be a good plan to discuss first the marantic thromboses; and here again we shall consider separately the cases which occur among children and those which occur among adults.

Since, as mentioned above, *thrombosis of the sinuses in children, accompanying conditions of the nature of marasmus*, is almost invariably the result of exhausting diarrhœas, which also give rise to cerebral anæmia, we should expect the symptoms, which the little patients present, to be essentially the same whichever of these two resulting conditions should actually be present; and such is indeed the fact. For a description of them, therefore, we may refer to the chapter on Hydrencephaloid.

In a given case, characterized by the symptoms there described, —collapse, followed by somnolence and coma, etc.—it might be doubtful whether we had to do with hydrencephaloid or thrombosis of the sinuses; for symptoms of this kind, due to severe disturbance of the sensorium, may occur also in connection with the latter affection. The clinical history of hydrencephaloid, however, usually closes with these cerebral symptoms, either in death or in recovery, and it is especially exceptional to meet with motor disorders, such as convulsions or paralysis, as a final result. The reverse is the case with marantic thrombosis. In the cases of this kind that have been described in detail, rigidity of the muscles of the neck has regularly been observed, sometimes also of the muscles of the back, and even of the limbs, and sometimes nystagmus.

In a few cases strabismus, ptosis, and paresis of the facial muscles have been seen.

It is true that symptoms of this same class are occasionally observed in connection with hydrencephaloid; this is, however, a very exceptional event, and we feel, therefore, justified in laying it down as a general rule, that when diarrhœas, occurring in children a few months old, are followed by cerebral disorders of the active motor kind just referred to, the diagnosis of thrombosis, and more particularly thrombosis of the superior longitudinal sinus, so often involved under such circumstances, is more probable than that of simple cerebral anæmia. At the same time it need hardly be said that these symptoms are in no wise pathognomonic, and do not justify an absolute diagnosis.

The general cerebral symptoms which accompany the thrombosis from marasmus, *i. e.*, venous stasis, in adults, are still more varied and indefinite. Sometimes, besides a slight degree of apathy and general depression, nothing abnormal is to be observed. The question might, to be sure, be raised, whether in such cases the occlusion was complete—indeed, in one case of the kind reported by Cohn (l. c., p. 156), it is expressly stated that "the canal is not entirely closed, still containing freshly coagulated blood." The clinical features usually met with are those common to the diffused cerebral diseases (in Griesinger's sense), and may vary widely in their individual characters and in their manner of combination. The mental functions invariably suffer: in some cases the impairment begins at the outset, and gradually goes on, through the stages of simple depression and apathy, to complete coma; in other cases the coma is preceded by delirium, occasionally of a maniacal character. Now and then the patients complain at the outset of headache, which may become unusually severe, and finally give place to loss of consciousness. Nausea and vomiting are also observed; but neither the vomiting nor the headache is as common as the other symptoms. The condition of the pupils is variable. Besides these mental disturbances, disorders in the motor functions are usually but not invariably present. These are sometimes of irritative character, such as strabismus, trismus, contractures, involving now one-half the body, now both legs or both arms, tremor or clonic convulsions of epileptic character, which may be limited to the separate limbs, or may involve the whole body, taking on the form of an epileptic attack; sometimes of the nature of paralysis or paresis. Such paralysis may be limited to the facial nerve, or to the motor-oculi, or may involve one-half or the whole of the body; sometimes both sets of symptoms occur in conjunction: thus the extremities on one side may be the seat of contracture, on the other of paralysis.

None of the above-mentioned symptoms, either in children or in adults, justify an absolute diagnosis, since they are common to cerebral processes of widely different kinds. In case of thrombosis of the sinuses, moreover, the influences which give rise to them are not always the same. If the group of symptoms observed in cases of anæmia and those observed in cases of hyperæmia be compared with those just described, it will be impossible not to recognize certain points of similarity between them. Both these conditions are in fact present in cases of marantic thrombosis, *i. e.*, from stasis. The anæmia is brought about through the influence of the underlying affections; the (venous) hyperæmia through the occlusion of the longitudinal and transverse sinuses, as will be seen from the simple consideration of the manner in which the venous circulation of the brain takes place. Further, the meningeal apoplexies and the affections of the cortex cerebri unquestionably play their part in bringing about the result, especially in causing the paralyses of one side of the body, and the irritative phenomena.

It would then be impossible to recognize thrombosis of the sinuses with certainty through the symptoms hitherto described, and it is, in fact, impossible so long as the case presents no other clinical features than those of this general character. In some cases, however, certain signs make their appearance, which, *when they are present*—for in a great majority of cases they are wanting—render the establishment of the diagnosis much easier and more certain. These symptoms are also due to circulatory disturbances, resulting from the fact that the *veins outside of the skull, communicating with the affected sinuses, become swollen,* in consequence of the obliteration of the latter, and thus serve to indicate the nature of the process going on within. These diagnostic signs may thus, from time to time, come to be of almost pathognomonic significance.

The sinus longitudinalis superior communicates directly with the veins of the nasal cavities, and, by way of the emissaria Santorini, with those on the upper surface of the skull. Considerable importance is, therefore, to be attached to the occurrence of epistaxis, as is shown, for example, in the case of a small child, reported by von Dusch, and the same may be said of a condition which has been observed by Gerhardt—and by myself as well—the presence, namely, of tensely filled vessels, running from the anterior fontanelle to the neighborhood of the temples and ears on both sides of the head; in the case of adult persons, to be sure, this condition might be but little marked, on account of the presence of the hair and the small size of the emissaria Santorini. Gerhardt is perhaps right in ascribing importance to the presence of cyanosis of the face, confined to the parts supplied by the venæ faciales anteriores; yet this opinion should not be adopted without further proof, and still less, that of E. Fritz,[1] that excessive perspiration of the skin on the forehead, neck, and breast, but not on any other part, is of especial significance.

The *sinus transversus* stands in communication with a small vein which traverses the mastoid process, and which, as Griesinger has pointed out, is of importance in this connection. Thus, in cases of thrombosis of this sinus, œdema, limited to the parts behind the ear, may make its appearance, through the agency of this vessel. This statement is confirmed by the statements of Mohs, perhaps also by those of Heubner. When, in connection with occlusion of one of the sinus transversi, the petrosus inferior is also involved, or (even without the latter) the vena jugularis interna, at its point of origin, the jugularis externa is enabled to empty itself, with more than usual freedom, into the interna (on account of the collapsed condition of the latter), and, consequently, will be found less well filled on the side of the neck corresponding to that of the lesion than on the other side. This diagnostic indication, however, of which Gerhardt was the first to recognize the significance, is, unfortunately, but rarely to be turned to account; for, in the first place, it happens only exceptionally that the position of the thrombus is that which has been described; further, in the case of enfeebled children, it is seldom that the amount of the blood circulating in the body is sufficient to render the external jugular veins visible at all (not to speak of recognizing a difference between the two in point of size); and, finally, in the case of adults, this vein is partially concealed by the subcutaneous fatty tissue. When both the sinus transversi are occluded, but not the superior longitudinal sinus—a condition which is but rarely met with—the same symptoms may be present as in cases of affection of the superior longitudinal sinus itself.

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[1] Gaz. Med. Hebd. 1857. No. 52.

The relations of the sinus cavernosus to other veins are such as to give rise, under the fitting conditions, to pronounced and characteristic symptoms. These were noticed by some of the earlier observers; but especial stress has been laid upon their diagnostic importance within the last few years, particularly by Corazza, Heubner, Huguénin, Genowille, and others. The ophthalmic veins are the ones through whose agency these symptoms are brought about. Thus, in case of thrombosis of the sinus cavernosus, venous hyperæmia of the fundus oculi has been observed, as well as œdema of the eyelids and the conjunctiva, together with prominence of the eyeball, due to hyperæmia of the retro-bulbar veins and of the vena frontalis. These symptoms may persist until the death of the patient, or they may disappear during life. In cases of thrombosis of the cavernous sinus, one important symptom may be present which is peculiar to the affection of that vessel, and is due to the fact that, in its walls and neighborhood, various nerve-trunks are disposed, which are liable to be irritated or paralyzed by the pressure of the thrombosis (or the swelling of the peri-venous connective tissue), an accident from which important symptoms must result; these nerves are, the first division of the trigeminus, the trochlearis, the abducens, and the oculo-motorius. In this way a paralysis of the motor nerves may arise, and, in a case reported by Lebert, there was neuralgia in the distribution of the upper branch of the fifth nerve, and a trophic disturbance of the eye, such as may be produced experimentally by section of this nerve.

In the presence of the conditions just described, which admit of being objectively recognized, and by a careful consideration of all the attendant symptoms in the case, it might certainly be possible to arrive at a diagnosis which would have more than probability in its favor. It is evident that all the symptoms due directly to venous stasis may be present as well in cases of phlebitic thrombosis as in those of the simple marantic thrombosis.

We must now return to speak again of the marantic thromboses in the case of infants. In consequence of the underlying affections in which this condition originates, the fontanelle is, regularly, yielding and depressed, and the edges of the bones pushed, sometimes, one over the other. Gerhardt has called attention to the fact that, in the course of the disease, the fontanelle may again become tense and prominent, the cranial bones pressed apart, and the sutures put upon the stretch. This event attends the occurrence of hydrocephalus from venous stasis, or of extensive meningeal or intracerebral hemorrhage, resulting from the thrombosis of the sinuses. This fact is worth bearing in mind, because through ignorance of it one might be tempted, in consequence of the above-mentioned change in the condition of affairs, to abandon the diagnosis of thrombosis, however probable it might appear for other reasons.

Let us now pass to a consideration of the *phlebitic variety of thrombosis*. Griesinger has pointed out that in these cases, as well as in those of the former variety, the thrombosis, as such, may, in virtue of the anatomical relations of the vessels involved, give rise to cerebral symptoms of certain definite kinds. Since, however, even the occlusion of both sinus transversi, which is of so common occurrence under these circumstances, does not cause so irremediable a disturbance of the circulation as that of the longitudinal sinus, these symptoms are likely to be less prominent in the former case than in the latter.

It is well known that otitis interna, or injuries of the head, may be followed by cerebral affections of widely different nature, the commonest being meningitis, purulent thrombosis of the sinuses, and cerebral abscess. The general symptoms due to these diseases may be identical with those which have been described as following the marantic form of thrombosis, in the case of adults. It must be, then, only through the presence of certain special characteristic signs that it will be possible to distinguish clinically between these three pathological conditions; and the difficulty of so doing is increased by the fact that the three affections are very often found conjoined. In most of the cases of purulent thrombosis that have come under our own observation, this complication was actually present. In one of the most recent, to be sure, the only cerebral lesion present was purulent inflammation of the transverse sinus, from otitis; but, as an offset to this fact, the cerebral symptoms were but little prominent, and a certain diagnosis was possible only when it had become evident that pulmonary embolism had occurred, which resulted in pneumothorax, which rapidly proved fatal. The only cerebral symptoms were headache and confusion of mind.

A comparison of the clinical histories of those cases, where suppurative thrombosis alone was found to be present, unattended by meningitis or other lesions of the cerebral substance, shows the following to be apparently its most important features (of course we leave out of account the symptoms due to the primary underlying affection).

In the first place, those cases are to be mentioned where the process remained latent throughout its entire course, the patients dying from other diseases, and the phlebitis sinuum only being discovered after death. This is, however, a rare occurrence.

As a rule, the clinical history is like that observed in cases of *septicæmia with especially prominent cerebral symptoms.* Sometimes the symptoms come on insidiously; at other times the attack opens with a chill, which is apt to recur frequently in the course of the disease. A state of remittent fever may make its appearance, in which case the patients have a characteristic typhoid look, with dry tongue, loss of appetite, and mental confusion. The cerebral symptoms, then, become constantly more and more prominent, and the depression of the mental faculties increases until it amounts to sopor or coma. In other cases delirium is present, which is usually mild in character, though sometimes exceedingly active.

Loss of consciousness usually comes on finally, and closes the scene. The motor and sensitive disturbances of other kinds, which often occur, such as paresis and paralysis, convulsions, hyperalgesia, and the like, are due to the accompanying meningitis and the lesions of the brain, and do not belong among the proper symptoms of thrombosis of the sinuses. (Compare the above-mentioned case.) Pain in the head also, especially unilateral pain, which certain observers—for example, Lebert and Cohn—regard as significant, is an inconstant symptom (Griesinger), and is probably never among the direct results of thrombosis of the sinuses, except when it accompanies inflammation of the cavernous sinus, arising in the manner described above.

Lebert lays stress upon an asserted tendency of the symptoms, in many cases, to become alternately more and less severe, remitting at times to such a degree that recovery seems possible, and then again suddenly taking on a more unfavorable character.

From a clinical point of view, then, the case stands thus: When a patient, who has received an injury to the head, or is suffering from caries of the internal ear, or from furuncles in the face, develops symptoms like those of pyæmia, with especial disturbance of the cerebral functions, the suspicion may be entertained that we have to do with purulent phlebitis sinuum, which is known to follow these, among other, conditions. (Vide under Etiology.) The correctness of this suspicion will become more than probable if disturbances of the circulation make their appearance, such as were described as resulting directly from the thrombosis of the sinuses.

Still one other important symptom is to be mentioned, which may accompany either of the two forms of thrombosis, the simple or the inflammatory, and which, when present, is of almost the same degree of significance with the immediate disturbances of circulation: and that is *pulmonary embolism.* Particles may be carried off by the blood-current from thrombi in the cerebral veins, just as from those in any other of the peripheral veins of the body, and become lodged in the lungs. If pulmonary embolism, therefore, is met with under conditions, and in connection with symptoms, which point to the existence of thrombosis of the sinuses, it becomes very much more probable, at times even certain, that this affection is actually present.

It would be, of course, impossible to enter here upon a discussion of the clinical history of pulmonary embolism in general, which is determined in part by the character of the embolus—*i. e.*, whether it is purulent or simply fibrinous—and in part by other circumstances.

Course—Prognosis.

It is difficult to determine the normal duration of the process, for the very simple reason that it is scarcely ever possible to fix the time at which the thrombus begins to form. The laying down of times is therefore a matter of guess-work. It can only be said, in general terms, that the case may stretch over several weeks, but is often brought to an earlier close, either through the accompanying meningitis, or (in the marantic form of the affection) through the underlying, exhausting diseases, death sometimes occurring even within a few days.

The prognosis is very unfavorable, the termination being almost invariably fatal. Recovery may, however, take place, even in the case of the marantic thrombosis of children; or, to speak more exactly, in view of the uncertainty which always exists as to the diagnosis, we are not in the position to deny that such is the fact. Indeed, isolated instances of recovery are reported, the reliability of which seems to be beyond doubt; such is one communicated by Sédillot (vide Lebert) and another, still more convincing, by Griesinger. These are of course exceptions.

Treatment.

It is plain that we can do nothing towards overcoming the vascular occlusion itself—supposing it to have become fairly established. At the most, a *symptomatic* treatment is all that can be employed to advantage, and this must naturally vary according to the requirements of the individual case, and often enough proves fruitless even where the diagnosis was correct.

By what conceivable means, for instance, in case of marantic thrombosis of the sinus longitudinalis, could we hope to further the escape of venous blood at the convexity of the brain, and thereby diminish the cerebral pressure?

Suitable *prophylactic treatment*, seeking to remove the conditions which might lead to the formation of the thrombi, is the only kind that is capable of being of service.

C.—Occlusion of the Cerebral Capillaries.

A series of experimental investigations have shown that the extensive occlusion of cerebral capillaries may give rise to even very marked disorders of the cerebral functions. It might therefore be expected that clinically also more or less serious cerebral symptoms would have been met with under similar conditions. This is in fact the case with one form of capillary occlusion, viz., that by pigment embolism, in which a large territory is regularly involved.

The whole process has, however, been but little studied, either from an anatomical or from a clinical point of view, and our acquaintance with it is as yet but fragmentary.

Etiology.

The best method of grouping the etiologically different varieties of capillary occlusion seems to be with reference to the varying construction of the thrombi or emboli. First in order, both because most important, and because best understood, come the *pigment embolisms.* The only circumstances under which this condition has been observed, when its existence was beyond a doubt, has been in connection with severe cases of malarial, intermittent fever. Since the affection has already been described at length in another part of this work,[1] we shall content ourselves here with this simple reference to the subject, and would remark expressly that it will be left entirely out of consideration in the following discussion.

The next variety of capillary occlusion to be mentioned is that through drops of fat. The origin of the obstructing masses may vary; in certain cases, without doubt, the fat is formed on the spot where the obstruction occurs; in others, it is swept thither from distant points. In the cases of the first order the source of the trouble lies in a degeneration of the arterioles and the capillaries themselves, of the nature of fatty degeneration (atheromatous degeneration), from which the cerebral vessels are especially prone to suffer. In the cases of the other variety, the fat may be carried into the brain from a variety of sources. The most common event is for the fatty contents of atheromatous formations, such as are found in the larger arteries, especially the aorta, to break through into the interior of these vessels, and thence be swept along by the current and lodged in the cerebral capillaries. Ebert[1] maintains that clots in the heart often give rise, by their decay, to fat-emboli.

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[1] Vide Vol. II.

Still another source of these emboli has been discovered within the last few years, through the investigations of E. Wagner, Busch,[2] Bergmann,[3] and others; this consists in injuries and inflammations of the bone; exceptionally in other processes. In case of injuries of the bones, the fatty tissue of the marrow is absorbed by the blood-vessels (and lymph-vessels), and gives rise, in the first place, to emboli in the lungs, sometimes only in them. In some cases, however, the drops of fat are small enough to pass through the lungs, lodging finally in still narrower capillaries, especially those of the brain, and blocking them up; it is nevertheless a rare event for fat-embolism in the brain to occur in this way. Since pulmonary fat-emboli originate elsewhere (for example, in purulent deposits) very much less often than in the medullary substance of the bones, these sources are of still less consequence for the production of cerebral embolisms.

Pus cells or white blood corpuscles may also occlude the cerebral capillaries. In case of pyæmic suppuration, this is sometimes accomplished by agglomerations of white blood corpuscles (as in cases reported by Cohn, for example). An observation of Bastian's is of interest in this connection, where, as the only cerebral lesion of importance, collections of white blood-cells were found in the small arteries and capillaries of the gray matter of the brain, in the case of a patient who had died in consequence of traumatic erysipelas capitis, with typhoid symptoms (delirium and stupor). At some points the cells constituting these masses were but few in number ; at others they amounted to two or three hundred, completely occluding the vessel in which they lay.

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[1] Tageblatt der Leipziger Naturforscherversammlung. 1872.

[2] Virchow's Arch. 35 Bd.

[3] Zur Lehre von d. Embolie. Dorpat, 1863.

Bastian suggests, on the basis of this observation, that thromboses or embolisms of this kind, affecting the vessels of the cortex, may be a frequent cause of the delirium and the other disturbances of the sensorium, occurring in various of the acute diseases attended with high fever. We must wait for further observations before undertaking to discuss this theory critically; the evidence afforded by isolated observations is of no consequence, either for or against the view.

The results of the investigations of other observers, where nothing abnormal was found in the cerebral capillaries, seem, as far as they go, to militate against Bastian's assumption. Thrombosis of the capillaries of the brain (also of those in the liver and spleen), through these masses of white blood-cells, was also observed by Feltz in the case of a leucocythæmic patient.

It is impossible to conclude, from the brief statement of Th. Simon's,[1] with regard to a case reported by him, that here and there an extensive new-formation of "lymph-elements" had taken place, whether or not this same condition was present.

Small particles from *fibrinous clots* are sometimes carried into the circulation and lodged in the cerebral capillaries. They originate in the same manner as do the larger emboli of the same kind, which were described above.

The temptation is great to refer the severe cerebral symptoms which occur in the so-called "cerebral" variety of acute rheumatism, to blocking of the capillaries with emboli made up of particles of fibrin from the valves of the heart. Against this view there are to be brought forward the negative results of the investigations undertaken with regard to this very point, and still more the fact that cerebral rheumatism may occur entirely unattended by disease of the valves.

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[1] Centralbl. f. die med. Wissensch. 1868. S. 835.

It happens as a very rare occurrence that diseased masses of so-called *specific* nature, gangrenous or cancerous, are carried in this way as emboli. Cases of this kind have been reported by Virchow and Lancereaux, characterized by the fact that, in connection with gangrenous processes in the lungs, an analogous condition was found in the brain.

Finally, still another variety of occlusion is to be mentioned, of very exceptional occurrence, which has been carefully described by Delacour,[1] and designated by Virchow as *lime-metastasis.* Here lime-salts are deposited in the walls of the very smallest vessels, to such a degree that they become finally absolutely blocked. Virchow is of the opinion that the lime is first absorbed from diseased bones, since in the majority of such cases he was able to discover evidences of such disease, such as caries, and the like.

Pathological Anatomy.

The occlusion of the capillaries is of course only to be detected with the microscope. The nature of the obstructing mass varies, and need not be described in detail here. The number of the affected vessels is subject to very great variation. The vascular walls are found to be normal where the case was one of embolism; while in cases of thrombosis by fat-drops arising from atheromatous degeneration of the arterioles and capillaries, they are altered in a manner which is now probably familiar to all. In case of lime-metastasis, according to Delacour's description, a resistance is felt to the knife in cutting through the brain, especially in the centrum semiovale and in the corpora striata; while, on the cut surface itself, small, rough prominences are to be felt with the finger, like the stumps of a beard. Lime-salts are found deposited in the walls and in the interior of the arterioles and the capillaries.

It is worthy of notice that the larger vessels, which, as in the case of the regular atheromatous degeneration (with or without calcification), are the first to be attacked, are found to be unaffected under these circumstances.

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[1] Gaz. des Hôpitaux. 1850. S. 107.

The nature of the *secondary changes in the brain, dependent upon obstruction of the capillaries*, varies according to the number of the affected vessels. If these are few, the circulatory disturbance will probably be completely compensated for; if they are numerous, structural changes occur which are analogous to those following occlusion of the large arteries, as might have been expected in advance. It has been proved experimentally that the first effect consists in an anæmia of the brain; and, later, the various stages of necrobiosis are run through, resulting in complete softening. This final condition is entirely analogous to that which was described in connection with arterial occlusion, except in one respect.

In the cases of the latter class, it is very exceptional to find more than a single focus of softening; while in the cases of capillary occlusion there are often a number, usually of small size. The so-called multiple cerebral softening, which is often met with among old persons, and for which it is, in many cases, impossible to discover a cause, is probably due, as a rule, to capillary thromboses, consequent upon fatty degeneration of the small vessels. In case the emboli are of specific nature, cerebral abscess, or even circumscribed gangrene, may result.

Symptomatology.

If we exclude from consideration the cases of pigment-embolism attending severe intermittents, which is the only variety of the affection with regard to which we possess observations of any number, it may fairly be said to be very difficult, if not impossible, to lay down a clinical history for capillary embolism. We know, to be sure, through the experiments of Feltz, Prévost, and Cotard, that extensive embolism of very fine particles may in animals rapidly induce death, resulting from the diffuse anæmia of the brain, such as follows the occlusion of all the cerebral arteries. No clinical analogue to this event has, however, been as yet observed; and in the few cases where obstruction of the capillaries has been discovered after death, the symptoms were of such varied character, that they are by no means to be used as the basis for definite clinical generalizations. We must content ourselves, therefore, with a few short statements.

If the embolic masses are not numerous they need not give rise to any cerebral symptoms whatever; this is known to have happened, for example, in certain cases of fat-embolism.

If, as occasionally occurs, in consequence of autochthonous fat-thrombosis, a district of considerable size is deprived all at once of its nutriment, the case may open with apoplectic symptoms, and run through the regular course taken by the localized cerebral diseases in general. (Vide under Occlusion of the Arteries.) As a rule, the symptoms are of a more general kind, such as accompany diffused cerebral disease: such are dizziness, often headache, nausea, trembling, and weakness in the extremities. Above all, however, mental disturbances are prominent, such as marked loss of memory, and other signs of mental decay.

Besides these, if the softening has occurred in the nucleus lenticularis or corpus striatum, in the pons Varolii, etc., certain definite symptoms of localized disease may be present.

It is self-evident that to make a diagnosis in cases of this kind is impossible, and that one only could be led by way of exclusion to suspect the presence of capillary thrombosis or embolism.

We have already spoken of Bastian's suggestion with regard to the possible relation between febrile cerebral symptoms and capillary occlusion.

The nature of the *course* and *prognosis* of the affection in question has already been essentially developed. The isolated embolisms are without significance. If they are extensive, disturbances result, whose course is the same with that of the corresponding secondary effects of the occlusion of the larger arteries.

There is of course no specific *treatment.*[1]

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[1] Since the manuscript was completed in November, 1874, we were unable to make use of the many experimental and clinical investigations which have appeared since that date, especially those relating to the localization of the cerebral affections.

JENA, *Feb.*, 1876.

TUMORS OF THE BRAIN

AND

ITS MEMBRANES.

OBERNIER.

TUMORS OF THE BRAIN AND ITS MEMBRANES.

Passing by the older literature of the subject, we would refer the reader particularly to its treatment by Wunderlich, in his Handbuch der speciellen Pathologie und Therapie, and by Hasse in Virchow's Sammelwerk.

Friedreich, Beiträge zur Lehre von den Geschwülsten innerhalb der Schädelhöhle. Würzburg, 1853.—*Durand-Fardel*, Krankheiten des Greisenalters. Würzburg, 1858, p. 194.—*Griesinger*, Archiv der Heilkunde. 1860, p. 51.—*Ladame*, Symptomatologie und Diagnose der Hirngeschwülste. 1865.—*Virchow*, Die krankhaften Geschwülste. 1863-1867.—*Rindfleisch*, Lehrbuch der pathol. Gewebelehre. 1873, p. 610.—An enormous number of reports of cases may be found in the compilations (*Annuske*), and year-books; we shall merely mention the following: *Stiebel, Jr.*, Journal f. Kinderkrankheiten. 1855.—*Gull*, Med. Times and Gaz. May, 1862.—*Lancereaux*, Archives Générales. 1864, p. 47 and p. 190. —*Immermann*, Berl. klin. Wochenschrift. 1865, p. 117, et seq.—*Sanné*, Gaz. des Hôpit. 1866, p. 141.—*Lebert*, Berl. klin. Wochenschrift. (Cerebral Aneurisms.) 1866, p. 208.—*Luys*, Gaz. des Hôpit. 1867, p. 105.—*Hitzig*, Untersuchungen über das Gehirn. Berlin, 1874.—*Nothnagel*, Virchow's Archiv. Vol. VII. p. 184. Vol. VIII. p. 420. The ophthalmological literature in connection with the subject may, for the most part, be found as follows: *Mandelstamm*, Gräfe's Arch. 1873. Part II. p. 39.—*Michel*, Graefe's Archiv. 1873, p. 59.—*Michel*, Archiv für Heilkunde. Vol. XIV. p. 57.—*Manz*, Deutsch. Arch. f. klin. Med. Vol. IX. p. 339.—*Annuske*, Graefe's Archiv. 1873. Part III. p. 165. Beiträge zur Symptomatologie und Diagnose der Kleinhirntumoren, by *A. Ferber*. Marburg, 1875.

Among the tumors with which we have to deal are to be reckoned all those occurring intracranially, which attain a certain size, and which interfere with the functions of the central organs of the nervous system contained within the cranium, by displacing or destroying them. We are concerned, then, not only with tumors of the cerebrum and cerebellum, but with those also of the pons and medulla oblongata. To this chapter belong, moreover, the new-growths springing from the meninges, from the blood-vessels, and from the inner surface of the skull. For the sake of convenience, merely, in order to admit of their description collectively in another place, we must here omit the consideration of gummata, echinococci, cysticerci, and of cerebral abscess.

Etiology.

The influences which give rise to tumors in other localities are those also upon which tumors of the brain depend. In consequence of hereditary predisposition, an abnormal hyperplasia takes place in the connective and epithelial tissues of the blood-vessels and their sheaths. In this hyper-production the original character of the affected tissue may either be retained, or it may become more or less altered by modification of the newly-formed elements and by changes in their relations to the connective tissue and vascular distribution.

There is no doubt that cerebral tumors occur more frequently in men than in women. Upon the basis of various statistics which have been made, the proportion may, in general, be taken as that of 10 : 6. This circumstance might be brought into connection with the fact that the male brain, being more actively exerted, is consequently more exposed to vacillations in its nutrition, which would favor an unequal development of the various elementary tissues of the organ. An explanation may also be sought in the pernicious influences to which men are exposed, and from which women are comparatively free. The greater immoderation of men in their pleasures and in the use of stimulants may be mentioned here in first order. Abusus spirituosorum gives rise to determinations of blood to the brain, which gradually become permanent, and may then afford the impulse to a localized abnormal proliferation.

Special reference must be made under this head to injuries of the skull, which men receive in the struggle for existence, more commonly than women. Blows and falls of all kinds are preeminently attributes of the stronger sex. This point in the etiology of cerebral tumors was formerly overlooked ; but, since it has been appreciated by Wunderlich, Hasse, and, above all, by Virchow, confirmations of the fact come from all sides that injuries of the skull are, without doubt, a cause, not only of hyperostoses upon its inner surface, but also of diseased growths in the meninges and in the brain itself. One would be inclined to assume that, in consequence of the osseous cicatrix, the circulation in the diploë, and, secondarily, in the dura mater, will be modified, and that thus a stimulus will be given for a pathological development; or that the mechanical concussion of the brain at the time of the injury must tear the delicate connections of the nervous apparatus, conducing to fatty degeneration, and in this way to proliferation of the neuroglia. However this may be, the tumors in question form very gradually at a place corresponding to the previous injury of the skull, and only in the course of years attain deleterious dimensions.

We shall merely refer to the fact that syphilis, echinococci, and cysticerci play an important part in the production of cerebral tumors. Still more important in this respect is the rôle played by tuberculosis. It may truly be said, that of all cerebral tumors the tubercular tumor is the most frequent, and that it is usually accompanied by tuberculosis of other organs. It is rare for it to occur primarily in the brain, and in this respect it differs from cerebral cancer, which is more apt to be of primary origin.

Pathological Anatomy.

Some of the tumors occurring in the brain are more or less peculiar to it, while others are similar in their composition to the neoplasms of other organs.

First Group.

Glioma (Virchow) is formed by proliferation of the neuroglia. In respect of color and consistence, the tumors are often difficult to distinguish from the normal brain matter. They are, for the most part, whitish, or, when the vascular development is abundant, they are reddish, and may be divided into hard and soft gliomata. The composition of the former approaches that of the fibromata, but yet it is only in the most highly developed forms that an intercellular substance with parallel fibrillæ is attained to. The cells are scanty, and usually have several nuclei. The soft gliomata contain more cells, which present various appearances, but are commonly small and deficient in plasma. In the often very friable matrix, networks of fibrillæ are present, in the points of intersection of which cells and nuclei are imbedded. When the matrix assumes a more mucoid character, the soft glioma resembles closely the *myxoma*, while a great increase in the number and size of the cells produces forms allied to *sarcoma*.

Glioma grows slowly, reaches a considerable size (as large as a man's fist), and leads to a fatal termination only after it has existed for many years. Traumata often give rise to these tumors; and when they occur in the course of the growth of one, they tend to hasten it. Retrogressive metamorphosis of the tumor by fatty degeneration, etc., is not impossible. An abundant vascular development (teleangiectatic form) leads to hemorrhages, which are difficult to distinguish from simple apoplexies. In addition to the minute examination of the margins of the apoplexy, we may be aided in such cases by the fact that glioma usually occurs in the white substance of the hemisphere where apoplexies are rarely seen.

Hyperplasia of the pineal gland seems to be formed essentially upon the same structural type as glioma. "It presents a solid, grayish-red, slightly lobulated or else smooth, round tumor, which may grow as large as a walnut or even larger. Upon section, the well-known tissue of the pineal gland is found gray and moist, with abundant vascular supply, and in old persons the sand-like bodies too are rarely absent. The histological elements are somewhat larger and firmer than in the normal." There can be little doubt but that most of these enlargements, which occur at every time of life, are of great significance for the condition of the brain. "They produce their effect partly by pressure upon the corpora quadrigemina, and partly by compression of the vena magna Galeni, which, in its turn, may easily give rise to hydrocephalus.[1]

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[1] *Virchow*, Lehre v. d. krankh. Geschw. II. 149.

Next to this condition of the pineal gland in the rarity of their occurrence are the following tumors, which, owing to the slight disturbances, if any, that they produce, are of more anatomical than of clinical interest:

Psammoma, or sand tumor, which has its prototype in the sand occurring normally about the pineal gland, must be distinguished from other forms of new-growths which have undergone calcareous degeneration. This form of tumor is to be referred to an inflammatory proliferation of the cellular tissue, in which the calcareous deposit takes place, sometimes in the shape of loose, mostly round, stratified granules, or by the formation of variously shaped small bodies, compactly infiltrated into the cellular tissue. They grow usually from the dura mater, and are situated commonly at the base of the skull, appearing as hard, hemispherical tumors, of the size of a cherry-stone, white in color and smooth on the surface.

Melanoma is a very rare pigmented tumor, which has its origin in the pigment cells of the pia mater. It does not reach any considerable size, but may be multiple.[1]

Neuroma, a genuine hyperplasia of the gray substance, may be found in the shape of small tumors, varying in size from that of a millet-seed to that of a pea, and situated on the ventricular surface,[2] in the white substance,[3] or on the outer surface of the brain.[4] Neuroma has hitherto been very rarely observed, and only in persons with some mental aberration, congenital or acquired. Simon convinced himself of the presence in these gray swellings not only of a central medullary striation, but also of a fine structure analogous to the normal cortical convolutions. We are not of opinion that these tumefactions are the organs upon which every kind of psychical extravagance depends, or else they would be much more common.

Hyperplasia of the anterior half of the *pituitary gland*, which is no doubt the analogue of the thyroid gland, probably seldom advances to a strumous tumefaction of any considerable size. A cystic formation may, however, take place here, driving asunder the sella turcica, and mounting up into the third and lateral ventricles. In this respect the case described by Zenker[1] is well worthy of notice.

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[1] Virchow's Archiv. Vol. XVI. p. 181.

[2] *Virchow*, Lehre v. d. krankh. Geschw. Vol III. p. 266.

[3] *Meschede*, Virchow's Archiv. XXXVIII.

[4] *Simon*, Virchow's Archiv. LVIII.

True cysts are not so common in the brain as was formerly supposed. It is here a question either of a portion of the posterior cornu of the lateral ventricle being cut off from communication with the remainder of the cavity,[2] or of hydrops cysticus glandulæ pinealis, or of hydrops septi pellucidi,—all of which are very rare,—or else of the more common and usually quite harmless cystic formations in the choroidal plexus.

For the sake of completeness, we may also mention the *ecchondroses of the basilar process*, which take their origin from remains of the cartilage of the synchondrosis spheno-basilaris, and usually pass on into mucoid softening.[3]

Cholesteatomata provide us with the connecting link between the above neoplasms and those in the following group. Their structure resembles that of epithelioma, consisting partly of hardened epithelial cells, partly of epithelial cells which have undergone fatty degeneration. These cells are arranged in concentric layers, and the tumors themselves are hard, pearly, and non-vascular, seldom growing as large as a walnut. They generally lie in some hollow at the base of the skull, or in some recess of the brain. They unite, as Rindfleisch says, "the structure of an epithelial carcinoma with the harmlessness of a wart or weal."

Second Group.

The *tubercular tumor*[4] is by far the most common of this category. It consists in a gray, yellow, or yellowish-white, hard tumor, which sometimes appears laminated upon section, and frequently grows larger than a hazel-nut. In its period of growth it is surrounded by a layer formed of softer reddish tissue, with numerous vessels and small, gray miliary tubercles.

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[1] Virchow's Archiv. XII. p. 454.

[2] Hydrocele of the Fourth Ventricle. *Virchow*, Krankhafte Geschw. I. S. 183.

[3] *Virchow*, Entwickelung des Schädelgrundes, 1857, S. 47.—*Luschka*, Virchow's Archiv, XI. S. 8.—*Zenker*, Virchow's Archiv, XII. S. 108.

[4] This name is to be preferred to "solitary tubercle," especially as the tumor is by no means always solitary.

Rindfleisch[1] has lately shown that a layer of fibrous connective tissue (with interweaving of the fibrillæ) is situated in the immediate neighborhood of the tubercular tumor, and that it is outside this again that the soft embryonal tissue, with its rich cellular supply, occurs. He consequently regards a portion of the cerebral tubercle as a fibroid. This amounts, perhaps, to what Virchow says:[2] "In not a few cases the fibrous portion of the neoplasm predominates, so that one is obliged to recognize a sclerosis tuberculosa, in which the cell proliferation does not attain any remarkable height." The tubercular tumor may become calcified or softened in the centre. The latter generally occurs to a limited extent, and leads in a few cases to the formation of atheromatous cysts.

It is evident that gummy tumors in their varied destinies may readily be confounded with tubercular tumors. According to Virchow, the following are the characteristics of the gummy tumor: Angular, nodulated, with an uneven surface, frequently in connection with the dura mater, surrounded by a broad zone of gelatinous substance, not prone to undergo softening.

The favorite position of the tubercular tumor is the gray substance, especially of the cerebellum. It is decidedly most prevalent in childhood. It is sometimes primary, and frequently multiple.

Carcinoma appears usually as primary fungus hæmatodes, originating either from the outer or from the inner surface of the dura mater. The tumor advances rapidly towards the bones, softens and pierces them, and then spreads on the external surface of the skull as a luxuriant growth (fungus duræ matris). The dura mater withstands the penetration of the tumor growing from its internal surface just as stoutly as it resists it when it grows from the outer aspect. The growth is then directed entirely towards the brain. We find communication between carcinomata within and without the dura mater only at pre-existing openings for nerves, such as the olfactory, optic, etc. Hence the growing in and out of cancers in the orbit, in the perforated plate of the ethmoid bone, in the spheno-maxillary fossa, and so on. Cerebral cancers are found sometimes as round, sometimes as very nodulated structures, and sometimes they occur flattened out, as upon the inner surface of the dura mater. Their color varies from a grayish-white to a yellowish-red, according to the amount of vascular development and the extent to which the retrogressive metamorphosis has gone. The microscope displays large cells crowded together in a scanty bed of blood-vessels, and altered glia fibres. Around the tumor is found a broad gray zone, of greater or less width, in which, besides disorganized nervous elements, an abundant vascular and cellular development may be seen.

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[1] Pathologische Gewebelehre. 3d Edition, p. 621.

[2] Die krankhaften Geschwülste, p. 659.

Sarcoma occurs in a variety of forms in the brain. Thus these tumors appear as hard, rather non-vascular, grayish-white, round, and somewhat nodulated formations, which, being well defined from the brain substance, admit of being easily removed entire. The latter may also be sometimes successfully accomplished with soft cellular sarcomata, which, however, in respect of their finer organization, present many transitions to other forms of tumor. It is therefore all the more necessary, with regard to these points, to refer to the text-books of pathological anatomy for a detailed description. We shall only in this place remind the reader that melanosarcoma is very liable to give rise to multiple metastases to the brain and its meninges.

Myxoma is rarer in the brain than in the rest of the nervous system. The occurrence of mucous degeneration in other cerebral tumors, however, is frequent.[1]

Lipoma is very rarely found in the brain, and, according to Virchow, may be most readily expected in the raphe of the corpus callosum and fornix, because there fat is most frequently present.

Osteomata—true formations of bone, not petrifactions, etc., of other neoplasms—have been observed so frequently in the brain, that one cannot doubt their occurrence. Osseous formations in the dura mater after traumata are more common.

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[1] In consequence of the intermingling of various stages of development, a great variety of cerebral tumors are produced, of which interesting instances are given in Rindfleisch's Pathologische Gewebelehre, with drawings. See also at page 610 of the same work for a classification of cerebral tumors founded upon their development.

Angioma also occurs as a tumor of the brain,—most commonly, indeed, as a complication of other tumors. We have already referred to it in speaking of some other tumors, *e. g.*, the teleangiectatic glioma. It is well, under any circumstances, to direct attention to this form of tumor, inasmuch as its occurrence in important centres, as in the corpus striatum, and on the floor of the fourth ventricle, gives it especial interest. To this class belong also the growths upon the inner surface of the dura mater, described under the name of pachymeningitis hæmorrhagica bregmatica.

Intracranial *aneurisms*[1] are not rare. They are of various sizes, and are found usually upon the large vessels at the base of the brain. They may arise in consequence of atheroma, or without this. Thus we observed an aneurism, in a man of thirty-four years, which was as large as a bean, and sat upon the arteria corp. callosi dextra at the genu. In other parts of the body, also, without any atheromatous disease, aneurismatic distentions of the vessels were found—amongst others, one of the size of a pea in the pelvis of the left kidney. Both the aneurisms just mentioned burst simultaneously in coitu, thus causing death. This case indicates the most common termination for cerebral aneurisms—namely, in fatal apoplexies.

We have so far considered the behavior of the brain, in relation to the various neoplasms, only in so far as those alterations which take place in the immediate neighborhood of the tumor have been referred to. There are, however, other alterations which gain significance according to the position of the tumor. Nervous trunks may be pressed upon or stretched. Important roads of communication in the central nervous system, even at considerable distances from the tumor, may become atrophied or undergo fatty degeneration.[2] Then, again, the microscopical examination of the brain generally shows that it has suffered from the gradually increasing pressure caused by the growth of the tumor. One frequently finds the surface of the brain dry, the convolutions, particularly upon the diseased side, flattened, the sulci shallow, and the blood-vessels empty. The parts lying in the mesian line suffer a displacement towards the sound side, of which the extent of course bears a direct relation to the growth of the tumor. The condition of the ventricles in these cases must, manifestly, be various. Sometimes they are narrowed by compression, but most commonly they are distended.

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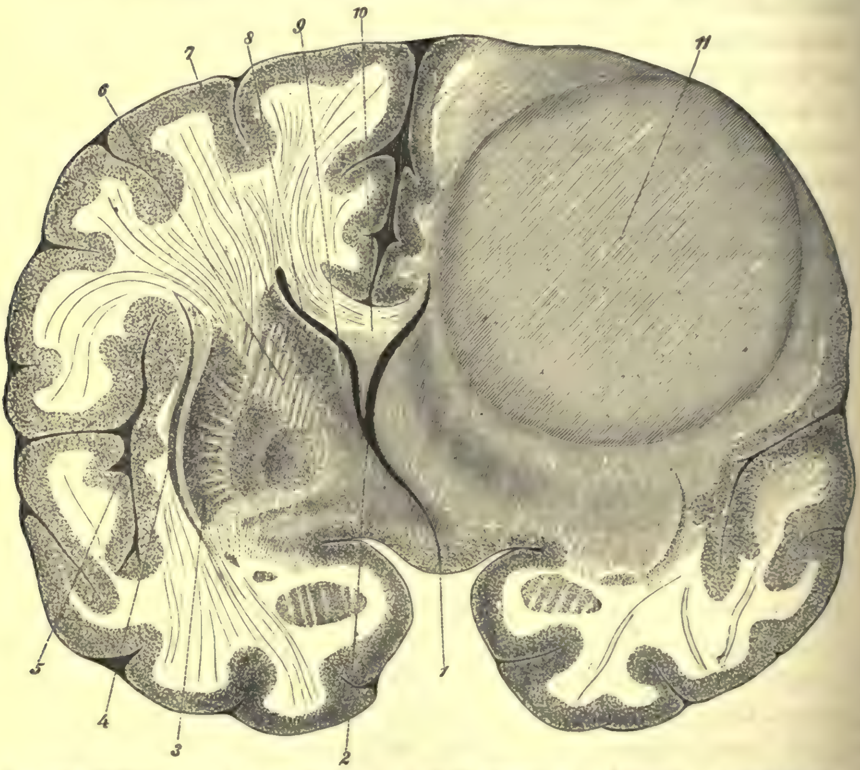
[1] For a full discussion of this subject by Lebert, consult the Berl. klin. Wochenschr. pp. 209, 229, 249, 336, 345, 386, 402.

[2] There is no doubt but that this point has been too little attended to in the records of autopsies.



1. Tuber cinereum. 2. Third ventricle. 3. Nucleus lentiformis 4. Claustrum. 5. Island of Reil.
6. Radiating fibres at base. 7. Corpus striatum. 8. Lateral ventricle. 9. Optic thalamus. 11. Tumor.

The preceding description of the anatomical changes will be made more clear by the accompanying geometric drawing (two-thirds of natural size), which was made from a most exquisite example of cerebral tumor. It represents the anterior surface of a perpendicular section of the brain, which, on the one side, passes through the greatest diameter of the tumor, on the other, through the end of the tuber cinereum. In explanation, we may briefly say that the preparation was taken from a woman of thirty-seven years. From her twenty-eighth year she had occasionally suffered from pain in the stomach, and headache on the left side of the head. Two years before her death she had a fall from a height of ten feet, after which she was unconscious for a short time without having suffered any injury to the skull. Subsequently violent pain in the left side of the head and vomiting came on. Fifteen months before her death she was attacked with violent fits, attended by unconsciousness and cramps, particularly of the right side, trismus, great depression, frequent weeping, increasing aphasia, complete paralysis of the right side, of the face and body, and, finally, general paralysis, coma, and death.

I. Symptoms in General.

Here the fact must, first of all, be brought to mind, that at autopsies intracranial tumors are sometimes discovered of which there had been no apparent symptoms during life. From this the conclusion has been drawn that cerebral tumors do not always give rise to disturbances of function of the organ. With the exception of the very small and of the very slow-growing tumors, this conclusion is incorrect; every tumor, of any size, which presses upon or injures the brain must give rise to symptoms, either transitory or otherwise. The degree of intensity of these symptoms depends upon a great variety of circumstances. In the first instance, individuality must be taken into account. It is known that many men react to the same irritation much more actively than do others; it is also known that there are phases in the development of the organism in which the most violent reflex cramps, after insignificant irritations, are common phenomena, while at other times the most severe irritations produce only slight results of this kind. And, again, whoever has observingly applied an electrode, knows that the same intensity of current will produce quite a different effect in different individuals, and even in the same individual at different times. Hence it follows that the symptoms consequent upon cerebral tumors vary greatly according to individual irritability. Furthermore, the power of accommodation of the brain is to be taken into account, that is to say, its capability of compensating for disturbances in the conduction of the brain once excited. Nothnagel[1] has lately shown that very slight injuries of the brain, by means of chromic acid, produce the most exquisite motor disturbances. No hemiplegia takes place, nor does the animal lose the power of moving itself, and only after careful observation one may perceive that the animal moves an extremity upon the opposite side of the body from that upon which the injury is situated somewhat awkwardly, that it does not retain its control over it. These disturbances of motion, however, pass off again in a comparatively short time, although the deposit of chromic acid in the brain remains, and the cerebral tissue at that place is permanently destroyed. Consequently, in the meanwhile, the conduction must have been undertaken by other paths,—the brain must have accommodated itself to the disturbance in its conduction. It is quite the same with cerebral tumors. They give rise, almost without exception, to symptoms, only that sometimes their efflorescence occurs at a period in which the patient may not have been observed or when other diseases disguised the symptoms of the tumor. The incipient stages of a cerebral tumor are, moreover, often doubtful and difficult to unravel, and it may easily happen that at an autopsy one is surprised to find in the cavum cranii a small fresh tumor, or an older but slowly growing one. But it should not therefore be supposed that the tumor which has been found has run its course without symptoms.

The power of accommodation is of course very different in different parts of the central nervous system. There are some centres in which comparatively little pressure, and others again in which not the slightest pressure can be compensated for. Thus lesions, which directly affect the origin of certain cerebral nerves, produce just as complete a paralyzing effect as certain lesions of the spinal cord produce upon the nerves of the extremities. Hence, taking into consideration the anatomical arrangement of the central nervous system in general, the conclusion may be drawn that its power of accommodation in respect of neoplasms, etc., is so much the greater the further the locus morbi lies from the base of the brain and from the cerebral ganglia towards the expanded surface (*Projectionsfeld*).

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[1] Virchow's Archiv. LVII. p. 184. LVIII p. 420.

The alterations in size of the tumor, and the rapidity with which they take place, have the most decided influence upon the intensity and kind of the symptoms produced by it; or, in other words, the intensity of the symptoms stands in a direct relation to the rapidity of growth of a cerebral tumor. Of course this growth need not always depend upon increase of the tissue elements themselves, but may be produced by hyperæmia of the neighboring parts, giving rise to a greater flow of fluids towards the tumor, and even cells and membranes, which have undergone a retrogressive metamorphosis, take part in the tumefaction by their swelling.

In the case of a young man, who had been under our care, a tumor, the size of a cherry, was situated in the frontal lobe. It did not give rise to any symptoms during the last few years of his life, which, however, ended with violent symptoms (convulsions, etc.), in consequence of alcohol and exciting living having induced determinations of blood to the brain. An increase in the tissue elements of the tumor itself had, in this instance, certainly not taken place, for these were already beginning to undergo retrograde metamorphosis.

It cannot be objected to the above that one sometimes finds an intracranial tumor, *post-mortem*, in old epileptics, for instance, who have suffered from epilepsy up to their death, the entire composition of which tumor shows that it is several years old, and, in respect to its elements and size, has not undergone any alteration. For, quite apart from the difficulty of deciding whether the latter point may not have played an important part in the continued epilepsy, we must refer to the fact that we see epilepsy, which has been caused by the irritation of a splinter of glass, continue, although the foreign body was long since reremoved. The reflex centres have "memory" for diseased excitations.

Another not unimportant point of view for the estimation of the symptoms of a cerebral tumor, is the mode of its growth. Let us compare the immediate neighborhood of a tubercular tumor with that of a carcinoma. In the latter there is a constant proliferation of cells and vessels, a constantly advancing invasion of fresh tissue; in the former a growth, comparable rather with inflammatory exacerbations, of a soft, richly vascularized embryonal tissue, in which arise the tubercles that cause the increase in size of the tumor. It cannot be doubted but that both modes of growth, *ceteris paribus*, must give rise to great varieties, not only of the cerebral symptoms, but also of the general constitutional disturbances—as, for instance, with regard to fever, its presence and degree.

Of importance, finally, for the occurrence of violent symptoms, are the metamorphoses which take place in the tissue elements of the tumor itself. And here we must take into consideration the determinations which give rise to retrogressive, *i. e.*, for the most part, reducing processes ex vacuo in the tumor; we must also remind the reader of those hemorrhages into the tumor which call forth symptoms of localized hyperæmia, amounting in some cases to the appearances of apoplectic attacks.

We have already referred to the pressure under which the brain suffers at the seat of the tumor, and have brought certain symptoms and the modifications they undergo into connection with it. The pressure effort of the tumor, however, is not herewith exhausted. Other organs, which are not embedded, like the brain, in a stiff, unyielding capsule, may escape the pressure caused by an increasing growth. In the case of the brain, every considerable diminution of the space it occupies must influence the entire organ. At first this increased intracranial pressure exhibits itself by displacement of movable substances. In this way the liquor cerebrospinalis passes in great quantities between the inner and outer sheaths of the optic nerve (Schwalbe), and by its pressure gives rise to those ophthalmoscopic appearances which we see so early and so constantly[1] present in cases of cerebral tumors.

Again, in consequence of the same pressure, the quantity of blood in the brain, and the rapidity of its circulation, will be altered; and, since the veins are chiefly pressed upon, more favorable conditions are evidently given for the secretion of the liquor cerebrospinalis. It will sometimes, then, be necessary to watch for symptoms of cerebral anæmia, as well as of hydrops ventriculorum.

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[1] *Annuske*, Graefe's Archiv. Vol. XIX. Part III. p. 165.

Meningitis, finally, may modify the complex symptoms.

A.—Disturbances of the Psychical Functions.

The opinions held concerning the constancy and intensity of these disturbances vary very much—less so, however, concerning the kind of the symptoms. For our part, we agree with those (Friedreich) who accept the occurrence of psychical disturbances as the rule rather than the exception. Unfortunately it has been too much the custom in psychological medicine to adhere closely to a well-defined schema, and to deny the existence of psychical disturbances unless these happened to agree with the group of symptoms corresponding to some distinct species of disease. If the psychical qualities of a patient suffering from cerebral tumor be analyzed, and a careful comparison made of the former with the existing mental condition, certain symptoms of mental disturbance will be discovered in most cases where the tumor is a growing one. There will be observed, in the first place, a diseased alteration of the so-called mental disposition, or of the mode of reaction of our most central (psychical) conductions. In cases where the cerebral tumor is of large size, the psychical reaction is retarded, and the disposition depressed; the patients are silent, care for nothing, and gladly withdraw from any intense mental stimulation. This seclusion and want of energy often increase to a continued melancholy. The patients are complaining, cross, and lachrymose.

In other cases one finds the power of reaction easily exhausted, the disposition changeable. A trifle will make the tears flow abundantly, and at the next moment something equally unimportant will win a smile from the sad face. The patients are quite powerless to resist the influence of external impressions, and in this respect remind one of the similar condition which exists in persons affected with the paralytic form of insanity. A prevalently gay humor is very rare with these patients.

Unmistakable disturbances of the intellect take place, especially with large tumors. The inability to work up and fix mental impressions, and to reproduce and combine former ones, develops itself parallel with the growth of a large tumor. The patients themselves, when reminded of their weakness of memory in regard to some recent event, are often unwilling to admit this, and plead a momentary indisposition or some other trouble as the cause of their forgetfulness. Sometimes the disturbance is characterized by the disappearance of certain fields of thought and certain ideas ; sometimes again there seems to be no dearth whatever of ideas, but the power of expressing them by the right word is wanting. This is the opposite of what Goethe puts into the mouth of Mephistopheles concerning the science of theology : "Denn eben, wo Begriffe fehlen, da stellt ein Wort zur rechten Zeit sich ein." If some one utters the desired word in the presence of such a patient, he will generally repeat it, and for the most part too with considerable alacrity. When, therefore, the strong peripheral irritation from the auditory nerve is added to the desire emanating from the gray matter of the brain, the obstacle to the central conduction is overcome and the combination of the word is rendered possible. In discussing this matter, we do not wish to encroach upon a subject, which will be treated of more thoroughly in another place ; but we must bring one fact prominently forward — namely, that the condition referred to, the *aphasia*, is constantly present when a tumor *is situated on the left side*, *in the region of the island of Reil* (*claustrum*), *and extends as far as the second or third frontal convolution.* There occurs here, then, for the conduction originated in the great convoluted surface of the cortical gray matter of the brain, a looping together, recurrently, of the conducting paths, the integrity of which is indispensable for speech. It has often been a subject of discussion upon what the preponderance of the left hemisphere over the right depends. Without wishing to go further into the question here, we may remind the reader that, even before we learned to name objects with words, we were wont to indicate them by pointing with the right hand; that we learned spelling by aid of pointing with the right hand; that, later in life, when we wish to give short commands, instead of explaining them by words, we often merely make a motion of the right hand; that, when we wish to express ourselves very distinctly, we accompany and illustrate our speech by motions of the right hand; and, finally, in place of spoken words, we so frequently make use of written ones, again by aid of the right hand. All these motions of the right hand, originating in the gray matter—and which we might call speech by the hand, and which at an early period of our life existed as speech—can only have their origin, according to the anatomy of the parts, in the gray substance of the left hemisphere. Impulses accordingly originate here which are habitually reflected to the motor nerves of the right hand. Is it then absurd to say that the conductions, which at a later period undertake the reflection of the same excitations from the gray matter to the special apparatus of speech, should follow, at least for a considerable distance, the paths which have already been employed for a similar purpose? Certainly not. Be this, however, as it may, so much is certain, that disturbances of conduction in the region referred to produce aphasia: how extensive these disturbances must be, however, is most difficult to define. At page 240 a woodcut has been introduced representing a large tumor, which has partly destroyed, partly displaced the frontal and parietal lobes (especially the anterior central convolution), as well as the nucleus lenticularis, claustrum, etc. For six months before death complete paralysis of the right side and aphasia had existed. With this case, in which there were such extensive evidences of disease, we would compare another case of aphasia, in which apparently only insignificant material lesions had taken place. A youth of nineteen years, suffering from well-marked pulmonary tuberculosis, became aphasic nine days before his death—appearances of meningitis and slight hemiplegia of the right side having previously set in. The autopsy showed two tubercular tumors—one the size of a bean, the other as large as a pea, situated close to each other, between the second and third frontal convolutions. They appeared somewhat on the surface, and pressed upon the corresponding gyri. Were they alone the cause of the aphasia? Certainly not. For, in the first place, they were of older date; and, in the second, the fresh meningitis tuberculosa had produced its deposit chiefly about the left frontal lobe and in its sulci and along the blood-vessels as far as the island of Reil, and the development of the aphasia was no doubt connected with the origin of this inflammation. Great care is consequently necessary where precise localizations are attempted.

Disturbances of the intellect increase more and more, of course, under the influence of the rapid growth of a tumor, of the destruction and modified irritability of the central comducting paths; mental action becomes constantly more limited, and imbecility establishes itself. Should somnolence and coma be added, one may expect that the final scene will be brought about by cerebral pressure and hydrocephalus. Besides this, to a certain extent, ordinary course of symptoms, there are always cases in which extraordinary disturbances appear. Sometimes the patients are affected with illusions—as, for instance, that in which they imagine themselves followed by some one who has an ill-will towards them (*Verfolgungswahn*)—or, more rarely, they imagine themselves to be great people (*Grössenwahn*). Again, maniacal states will occur, now and then, in the course of the disease, or derangement of the mental organs may display itself from beginning to end.

Hallucinations are more uncommon than one would perhaps be inclined to suppose, and they rarely have any value as an aid in the diagnosis.

On the other hand, it is worth calling to mind that Friedreich observed excessive sleepiness to be a chief symptom in a case of cerebral tumor. We have also observed the same thing in a case of glioma of the parietal and occipital lobes.

B.—Disturbances of the Nerves of Sense.

The olfactory nerve, which is situated most anteriorly, will be chiefly injured by tumors at the base of the brain, in front of the optic chiasma. Inasmuch as the sense of smell is not very well developed in man, and varies in its acuteness in different individuals, and inasmuch as disturbances of it, in consequence of habitual colds in the head, ozæna, polypi, etc., are very common, weight can only be attached to a monolateral or double-sided disturbance of this sense, when the ordinary causes of it can be excluded and other symptoms of cerebral tumors are present.

Of much greater import for us is the condition of the optic nerve. We have here amblyopia and amaurosis, as the cause of which the ophthalmoscope shows (A. von Graefe) the choked disc or congestion papilla (*Stauungspapille*) and neuroretinitis, and attention must be specially directed towards contractions in the field of vision.

Decrease in the acuteness of vision, even complete blindness, is not only very frequent in cases of cerebral tumors, but also often one of the first symptoms. Upon the basis of a very diligently collected, complete compilation of cases, Annuske[1] arrives at the conclusion "that optic neuritis is almost without exception a constant attendant upon cerebral tumors, and consequently occupies the first rank among all the symptoms of intracranial neoplasms."

So long as the absence of the choked disc in cerebral tumors is regarded as a rare occurrence, as other ophthalmologists also consider it to be, one cannot value the importance of the symptom in question too highly, nor be insensible to the duty of subjecting "suspicious disturbances of sight" to a careful ophthalmoscopic investigation. It does not concern us here to enter into a description of the ophthalmoscopic picture, to paint the swollen, opaque, and imperfectly defined optic papilla, with its fine arteries, and broad and tortuous veins; it is of more importance for the comprehension of the appearances, to remark that we have to deal here with a degenerative process in the nervous elements, in which the increase of the intracranial pressure plays a chief part.[2] If, namely, in consequence of the development of an intracranial tumor, the pressure within the cavity of the skull be increased, the fluid between the sheaths of the optic nerve, which communicates (Schwalbe) with that in the subdural space, will become dammed up, and the optic nerve at its place of entrance into the globe will become incarcerated, and will undergo an œdematous swelling at this point, which, no doubt, hastens disorganization in the prolongation of the nerve.[1] Of course, this form of disturbance of vision, one of whose characteristics is that it affects both eyes simultaneously, although perhaps not to an equal degree, is not the only kind which is found. Lesions in the region of origin of the optic nerve, as well as interruptions in the tractus opticus and optic chiasma, carry with them, naturally, the most serious consequences for vision. It is chiefly when tumors implicate the tractus opticus and chiasma, that contractions in the field of vision and bilateral hemiopia make their appearance ; and these symptoms are most important for us, for it is by a precise investigation of them, combined with a consideration of any existing disturbances in the functions of other cerebral nerves, that we may succeed in localizing tumors at the base of the brain with accuracy. It is in this respect worthy of observation that later investigations of Mandelstamm[2] and Michel[3] have shown a complete decussation of the optic nerve-fibres in the chiasma, and in consequence of this the diagnostic interpretation of the various forms of hemiopia—of which we shall speak further on—has considerably gained in accuracy. Moreover, in the interest of the differential diagnosis, we must not omit to refer to what Michel says with regard to the changes which take place in the recessus,[4] which penetrates from the surface into the optic chiasma, and communicates with the infundibulum and with the cerebral ventricles. "As soon as a quantity of fluid has collected in the ventricles, in consequence of the existing communication, it will pass into the recessus above the chiasma ; the amount of fluid present in the recessus will then simply depend upon the quantity of the fluid present in the ventricles, and upon the pressure under which it rests; and then again, in turn, the interruption in the functions of some or all of the fibres of the optic nerve will be more or less complete in proportion to the amount of fluid present in the recessus and to the pressure to which it is subjected."

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[1] Graefe's Archiv. 19. Vol. II. Part, p. 165.

[2] *Manz*, Deutsches Archiv für klin. Med. Vol. IX. p. 339. *Michel*, Archiv für Heilkunde. 1873. XIVth year, p. 39.

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[1] In what manner the changes in the prolongation of the optic take place, is a question upon which authors are not agreed. That the choked disc may go back in consequence of retrogressive changes in the intracranial tumor, is, on the other hand, generally admitted.

[2] Graefe's Archiv. Part II. p. 39.

[3] Graefe's Archiv. Part II. p. 59.

[4] loc. cit. p. 78.

Michel then relates the case of a child, eleven months old, in which, in consequence of a high degree of hydrocephalus internus, the recessus was greatly distended, and contained a quantity of fluid. According to the same author, an amaurosis, occurring suddenly and in both eyes simultaneously, or quickly following upon each other, may be referred to the condition mentioned above. The functional disturbance may be subject to considerable fluctuations, and may even quite disappear under certain circumstances. The post-mortem examination, he says, furnishes for the most part only a negative result.

Subjective sensations of light are not rare, and *diplopia* belongs to the more common symptoms of cerebral tumors. It makes its appearance of course most constantly with tumors situated at the base of the brain, which encroach upon the origin or course of the third, sixth, or fourth pair, and is then usually the forerunner of persistent strabismus.

The *pupil*, in cases of developed intracranial tumor, is usually dilated, often very much so, and reacts but feebly to light. In many cases the pupils are of different sizes in the two eyes.

Disturbances in the *sense of hearing* are rarely to be regarded as genuine symptoms of cerebral tumors; they occur otherwise so frequently. And even complications, with disturbances of conduction in the region of the facial nerve, cannot be taken seriously into account, owing to the peculiar course it runs and the various accidents to which it is exposed on its way. But if other tumor symptoms should also appear, particularly paralyses of other cerebral nerves, then diminution or loss of hearing, or even tinnitus, in one or both ears, may become of considerable value for the diagnosis.

Alterations in the *sense of taste*, particularly unilateral ones, are present, without doubt, in some cases of cerebral tumor, although they often are difficult to define distinctly. We have at present under observation a case in which, besides altered sensation of the left side of the face, a distinct sense of taste seems to be wanting in the left side of the tongue; at the same time the unfortunate individual is so apathetic and out of temper, that we must admit the possibility of his standing the various tests of taste with another result, were he capable of greater attention. All kinds of subjective perceptions of taste, such as metallic, bitter, etc., are here very common, as in all affections of nerves.

Hyperæsthesia and *anæsthesia*, in cases of large intracranial tumors, but especially where they are growing rapidly, belong to the most common symptoms.

Headache must be mentioned first in this connection. For the most part it occurs all the more constantly the quicker the tumor grows and the closer it approaches the meninges. Its violence varies from a sensation of weight and tightness in the head to the most insupportable paroxysms of pain. Remissions of the pain are generally experienced, and occasionally may even assume a decided intermitting type. In cases where frontal headache prevails, it frequently occurs that, if asked to localize the position of greatest pain, the patient indicates the precise spot of the skull under which at the autopsy the tumor will be found. Sometimes also such a spot may be discovered by its great sensitiveness upon pressure and percussion, as also in coughing and laughing.

Closely connected with headache, are other forms of hyperæsthesia, which form the transition to the so-called anæsthesia dolorosa. We meet here, then, with the combination between *irritability* and *paralysis*, which we shall find again upon the motor territory.

In this connection, the trifacial nerve claims our first attention. It is frequently the seat of painful affections, which have nothing to do with cerebral tumors; but when all three divisions are simultaneously affected, the possibility of an intracranial tumor existing should be all the sooner taken into consideration. And this possibility will be increased to probability if, on the painful side of the face, sensation can be shown objectively to be diminished, and, in addition, if disturbances of other cerebral nerves are present. The intensity of the facial pain, which is chiefly a symptom of tumor at the base of the brain, is of course very variable, according to the special relation of the neoplasm to the nerves in each case. The quality of the sensation varies also; and besides the distinctly painful paroxysms, there may be feelings as if the part were swollen, numbness, formication, itching, etc.

In other parts of the body, too, anæsthesia proper is ushered in by abnormal sensations in the corresponding side of the body. Numbness, wandering pains, and formication, alternate with each other, before a distinct diminution of the power of feeling becomes evident, but they do not always cease even when this has become established. Hyperæsthesia and anæsthesia occur almost always upon the opposite side from the tumor; those only which lie quite close to the foramen magnum may make an exception to this. Neoplasms, occupying the cerebellum only, seem not to produce the usual disturbances of sensation; but they affect the so-called "muscular sense" all the more. An uncertain staggering gait belongs to the most constant symptoms of cerebellar tumors. We had under observation for a length of time a patient, aged thirty-seven years, in whom, besides well-marked symptoms of pulmonary tuberculosis, there was intense pain at a spot situated below and to the left of the protuberantia occipitalis externa, but with this exception there was no disturbance of sensation. The patient tottered greatly when he stood, and, upon closing his eyes, he fell at once. The autopsy demonstrated, in the left hemisphere of the cerebellum, a tubercular tumor the size of a pigeon's egg, which was attached to the dura mater for a considerable extent.

Hyperæsthesiæ in the sphere of the sexual appetite have also been brought into connection with neoplasms in the cerebellum. There have indeed been cases enough of such disease in which excesses in venere, or an insatiable propensity for masturbation, have been noted. At the same time, how often has this symptom been wanting with cerebellar tumors, and how often are such tumors absent while these symptoms are present!

Vertigo belongs to the most frequent, and often to the earliest, symptoms of cerebral tumors, and appears with varying intensity. Sometimes the sensation is that of falling forwards or backwards, sometimes that of rotatory motion. There is no doubt that these different forms of vertigo can be brought into relation with either irritation or paralysis of certain districts of the central nervous system ; but it is very difficult, in a given case, to indicate the precise region in which the lesion is situated. This depends upon the alterations in respect of irritability and blood supply, which an intracranial tumor can give rise to, not only at its seat, but also at a great distance off. In case of cerebral tumor, vertigo presents itself either in the form of transitory attacks, which recur in shorter or longer intervals, or it may be continuous, so that the patients cannot walk without tottering.

C.—Disturbances of Motion.

Cramps, or *epileptoid* attacks, are among the common symptoms of cerebral tumors. The seat and size of the latter determine the occurrence of these symptoms in a less degree than one might be inclined to suppose. The irritations to which the neoplasm gives rise, accumulate and are discharged—whether directly or indirectly by means of the vaso-motor nerves, is of no consequence here—in the form of clonic and tonic contractions of all the different groups of muscles during an interval of unconsciousness. In the absence of other symptoms, we regard such an attack as a simple epileptic fit. But the picture presents itself in another light, when the cerebral tumor, which has given rise to the fit, is large or occupies positions in the central nervous system, which represent special centres of motion.

In such a case it may be found that these cramps make their appearance, for the most part, unilaterally, and in special groups of muscles, and that in these same groups of muscles, particularly after the fits, paralysis gradually appears. Here, then, we have again the combination of irritation with paralysis. It is, however, by no means true that the cramps run always in the established groove of an epileptic fit. The most different hyperkineses come under notice ; and sometimes here and there, in distinct groups of muscles, an isolated, convulsive motion makes its appearance, like a flash of lightning, after which equally limited clonic or tonic contractions follow at one time, while at another a tremor will continue for hours in the same motor region.

With regard to the different groups of muscles which may become the seat of irritation or paralysis, it is all the more important to define them accurately, inasmuch as it is chiefly upon the basis which they afford that conclusions as to the position of the neoplasm are arrived at. Anatomico-physiological considerations—to all of which it would be impossible for us to refer now—are here, of course, the criterion. We shall consequently confine ourselves to the following observations: The paralyses consequent upon cerebral tumors are mostly hemiplegiæ, and in respect of their intensity and progress, they present by no means a constant relation. Often when their presence is but slightly indicated, they become, in the course of a night, very much increased, and often, when apparently undergoing a cure, they become more pronounced again after an epileptoid or apoplectoid attack. In fact, such an attack is sometimes nothing else than a true apoplexy; in consequence of retrogressive changes in the tumor a hemorrhage has taken place within it. Of course the paralyzed muscles, after such events, go through very much the same changes as those which are the result of a continued rest; they gradually atrophy. Nevertheless, their irritability for both kinds of currents remains for a long time, and only in very old cases does it display a distinct diminution. A fact, too, worthy of note is, that permanent contractions are by no means common.[1] The reflex activity exhibits no peculiarities which can be utilized for our purpose. Paralysis of the face lies very frequently upon the same side as that of the body, which would, accordingly, localize the tumor in the opposite hemisphere.

In other cases an *alternating paralysis* is present in face and extremities, and then the facial muscles, in respect of their irritability, may behave like peripherically paralyzed muscles; while in other parts of the body, in paralyzed regions, almost normal irritability prevails. This condition of affairs indicates that the neoplasm has injured the cerebral nerves in their course, but the motor fibres of the extremities intracerebrally, consequently above the decussation. The tumor lies, therefore, upon the side of the facial paralysis, at the base of the brain, not far from the middle line. The condition of the third, fourth, fifth, sixth, and seventh pairs of nerves, which arise from the brain, one behind the other, determines still more accurately the locality of the tumor.

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[1] They are most common when the corpus striatum is the seat of the neoplasm.

Paraplegia is a symptom of those tumors which either occur in the middle line, or are multiple, or attain such a size that they, either directly or indirectly, affect both hemispheres. We therefore have to reckon paraplegia among the other terminal symptoms of simple tumors. Paralysis of the bladder goes hand in hand with paraplegia.[1]

Functional disturbances in individual groups of muscles in the face and in the extremities demand our attention all the more, from the fact that Fritsch and Hitzig have lately endeavored to demonstrate centres of motion in all directions on the surface of the brain, both experimentally and by means of clinical observation (Hitzig); and for this purpose the records of cases of tumors occurring in the cortical substance have been appealed to for aid in elucidating the theoretical and practical questions still at issue on this subject. We are at present unable to adopt the proof which Hitzig[2] has offered by aid of a clinical case, and with all reserve, for the motor points upon the cerebral surface. We will give our reasons for this in a few words, referring at the same time to the case alluded to, because just here such an analysis is doubly in its place. The patient, in Hitzig's case,—a French soldier, aged twenty,—was grazed by a musket-ball upon the right side of the head, on the 10th of December, 1870, at Orleans. Subsequently the wound took on a bad appearance; and later on—on the 3d of February, 1871—without any symptoms worth naming having been observed, the bone was found to be bare at a point from five to eleven centimetres above the external auditory meatus. Some healthy granulations were present in the neighborhood. On the 4th of February—that is, about two months after the injury—cramps made their appearance in the region of the left side of the face, in the tongue, in the sterno-cleido-mastoid, in the anterior muscles of the neck and the respiratory muscles, and in the orbital muscles, etc. ("there were others which did not admit of investigation"), which were often repeated, with various modifications, and were accompanied by partial paresis of the facial and hypoglossal nerves. On the 10th of February the man died. At the spot mentioned above, where the bone lay bare, the autopsy demonstrated necrosis of a portion of the external table, four by two centimetres in size, with a well-marked line of demarcation all round. The bone at the corresponding part of the inner surface was of an abnormal color, and a bit of the vitreous table, as large as a lentil, was found almost quite detached, and bathed in pus. Precisely opposite to this point lay an abscess, the depth and size of the opening into which amounted to one and a half by two centimetres. In its neighborhood the cerebral substance was soft and the veins contained pus. The whole right hemisphere down to the base was of a blue-gray color, while the left hemisphere was red. The pia mater and dura mater were covered with thick pus. In the right hemisphere there were numerous minute hemorrhages. These were the most important points of the case. There are two epochs in its history: the first, from the day of the injury, 10th of December, to the 3d of February, during which, notwithstanding a wound of the skull, which had considerably injured the bone, no cerebral symptoms are noted; and the second, in which those symptoms set in with great violence, and ended in death within six days. In the same way we find in the brain two different conditions, corresponding to the course of the symptoms: the cerebral abscess, with progressive destruction of the surrounding brain substance consequent upon the injury, which remained latent until the 4th of February; and a severe purulent meningitis, the result of the abscess, which chiefly affected the right hemisphere, and ended fatally on the sixth day. Hitzig, however, does not arrive at the same conclusion. He is inclined to bring the localized symptoms of irritation and paralysis into connection with the abscess and its position, regarding the latter as the cause of the former, and employs the case as a proof that the centres for the affected territories are to be sought in the neighborhood of the abscess. Having said thus much about this case, let us pass on to the experiments of Fritsch and Hitzig, the importance of which, for the function of the individual parts of the brain and consequently for us, is not to be doubted. Both experimenters have found that in dogs, monkeys, etc., there exist comparatively circumscribed points, on the cerebral surface, of which the most localized possible[1] irritation calls forth certain motions in certain groups of muscles, and of which the extirpation produces paralysis in the same motor territories. We cannot of course enter into a description of the regions in question, but yet we would not like to omit laying before the reader a representation of their position. We consequently give a copy of Hitzig's drawing of a monkey's brain, with the points in question marked upon it.

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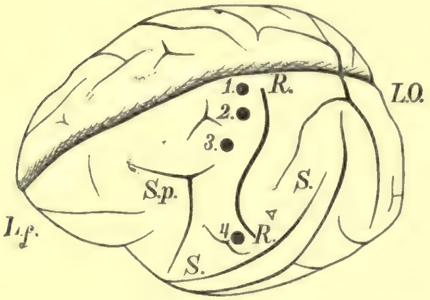
[1] Disturbances in the function of the bladder are generally observed in conjunction with neoplasms at the base of the brain and in the cerebellum.

[2] *Hitzig*, Untersuchungen über das Gehirn, page 115.



L.f. Frontal lobe. *L.O.* Occipital lobe. *S. S.* Fossa Sylvii. *S.p.* Sulcus præcentralis. *R. R.* Sulcus Rolando. 1. Centre for the hinder extremity. 2. Centre for the anterior extremity. 3. Part of the centres for the facial motions. 4. Centres for the motions of the mouth, tongue, and jaws.

It may be objected that these facts, obtained by observation upon monkeys, cannot be transferred to the human brain. Hitzig has disarmed this objection as far as possible. It is, indeed, sufficient to cast a glance at the surface of the human brain, in order to recognize the analogous positions of the same, and to arrive at the conclusion that the ascertained points in the monkey's brain would in the human brain fall in the neighborhood of the anterior central convolution. To simplify this, we insert also Hitzig's drawing of the human brain (after Ecker).

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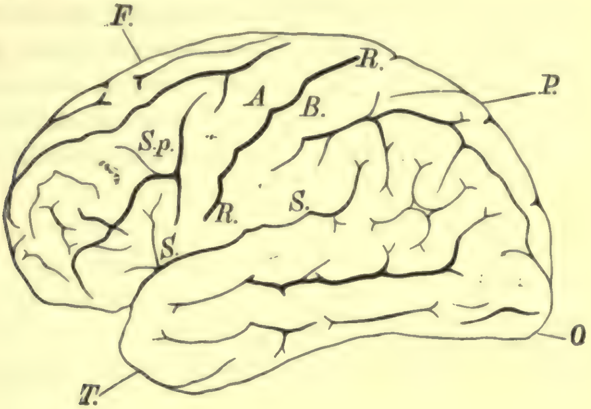
[1] Might not the localized irritation produce contraction of those vessels which pass deep into the brain, and thus transmit the irritation to other territories of conduction? Vide also concerning the experiments and deductions of Hitzig, L. Hermann's communications disproving them in Pflüger's Archiv, Vol. X., p. 77.

The lettering has the same significance as in the preceding figure. *A*, anterior central convolution; *B*, posterior central convolution.



It is evident that clinical observation may contribute very materially towards arriving at a decision with respect to these facts, which are of such consequence for cerebral pathology. The result, however, of the investigations previously made by Nothnagel does not inspire us with too sanguine hopes in this direction. Nothnagel produced disturbances of motion in rabbits by making deposits of chromic acid at points analogous to those of Fritsch and Hitzig, and thus destroyed the corresponding parts. The disturbances agreed partially with those observed by the latter authors; but they were not permanent, disappearing again after fourteen days. Hence, it would seem that in these centres it is a question of certain associations of motor paths for impulses arising in the gray matter, whose function, in case of their destruction, may be undertaken in some degree by other conducting paths which have remained intact. The practical deduction to be made from this is, that our observations for the purpose of localizing central nervous diseases must be as constant as they are accurate.

Finally, we have here to refer to certain less frequent disturbances of motion, the so-called compelled movements—forward, backward, in a circle, and rotatory movements. As the reader is aware, such motions may be produced in animals by injury to certain parts of the brain. Thus, Nothnagel has lately discovered a nucleus cursorius in the corpus striatum, upon injury of which animals rush on in an incessantly forward motion. Such compelled forward movements have occasionally been observed in man. We have ourselves seen a boy, who did not suffer from any localizable cerebral disease, but who after each epileptic attack gathered himself quickly together and hurried along as though in a wild chase for from three to five seconds. Compelled backward movements have been more frequently observed in affections of the cerebellum. They were present in the case, already referred to above, of a tumor the size of a pigeon's egg in the left hemisphere of the cerebellum. When the patient stood upright, he began immediately to put one leg behind the other at a quick rate, which usually resulted in his falling.

With regard to the so-called circus-movements, we are taught by experiments on animals that they are set up by the incision or extirpation of the pedunculi cerebri. The animals move in an arc, the convexity of which lies towards the side on which the pedunculi are injured. Equally well known are the rotatory movements, which division of the crura cerebelli ad pontem give rise to, and which are performed towards the paralyzed side. It is supposed in these cases that the muscles of the non-paralyzed side are always attempting to re-establish equilibrium, but, for want of assistance from the paralyzed side, a curvilinear or rotatory motion is brought about. Under any circumstances, we must bear these facts in mind in attempting to establish the symptomatology of cerebral tumors; but it would be very rash to draw the conclusion, for example, from the occasional occurrence of the so-called circus-movements, that a tumor occupies the pedunculi cerebri. The same motions have been seen with tumors lying deeply imbedded in the hemispheres, and have been wanting in some which were situated in the peduncles.

D.—Other Disturbances.

Vomiting occurs frequently with cerebral tumors, and is apt to be associated with attacks of vertigo and headache, and may be so obstinate that the nutrition suffers in consequence.

The bowels are generally costive in the final stage; but sometimes the motions pass involuntarily. Towards the close of the scene there is also trouble in emptying the bladder, the act being performed either with difficulty or without the patient's knowledge.

In rare cases the secretions display abnormalities. A flow of tears with lesion of the trifacial, and inosit in the urine with tumors of the fourth ventricle (Schultzen), are among the better known phenomena.

Disturbances of the circulation are observed only when the position or rapid growth of the tumor, or complications, produce an effect upon the pneumogastric. In the case of a quickly growing tumor in the parietal lobe, we saw for a length of time every exacerbation of the headache associated with a marked reduction in rapidity of the pulse. On the other hand, an increase in rapidity of the pulse belongs to the final symptoms.

With benign tumors a considerable diminution in the nutrition is often absent, and may even be quite wanting in cases of cerebral carcinomata. Frequent attacks of headache, vomiting, and cramps, however, usually cause the freshness of the complexion to vanish, and give the face a sallow color, which, especially in conjunction with facial paralysis and the usual pained expression of countenance, indicates a severe disease. The nutrition is affected most in cases of tubercular tumor; although even here exceptions are seen, particularly in children. Fever is certainly not the rule in the ordinary course of cerebral tumors. It is found, perhaps, most commonly in the incipient stage of tubercular tumor. As a terminal symptom, fever is not rare with cerebral tumor.

Finally, we have to mention the perforation which is liable to take place with intracranial tumors. It is most frequently seen with the so-called fungus duræ matris. The tumor perforates the skull, and then proliferates further upon its outer surface, and may exhibit the pulsating motions of the brain imparted to it. Pressure upon it sometimes gives rise to severe cerebral attacks, sickness, vomiting, fainting, convulsions, etc. The fungus duræ matris, however, is not the only growth which appears extracranially, but, as already mentioned, tumors which have originated on the inner side of the dura mater may extend through the foramina for the optic nerve, trifacial, or olfactory, into the orbit, antrum of Highmore, or nasal meati, respectively, and from thence give rise to all kinds of complicating symptoms.

II. Special Groups of Symptoms.

A.—According to their Course.

1. *Initial symptoms*, which rarely admit of a certain conclusion with respect to the existence of a tumor.

Uneasiness; ill-humor and irritability; forgetfulness; want of energy.

Subjective sensations of light; momentary diplopia; diminution of vision; commencing choked disc; tinnitus aurium.

Headache, sometimes continuous, sometimes intermitting, sometimes remitting; darting pains; formication; numbness, particularly when these symptoms occur on one side only.

Weariness, coming on with slight provocation.

Vertigo of varying degree.

Faintings; epileptoid attacks.

Vomiting.

2. *Symptoms which are peculiar to the developed cerebral tumors, and make good its diagnosis.*

Besides the appearances described as initial symptoms, which, as a rule, increase, we have here to mention the symptoms specially due to a localized cerebral trouble (*Herdsymptome*), which, in the case of cerebral tumors, are all the more characteristic, the more gradually they become developed and the more surely allied forms of disease can be excluded.

Depression of spirits, amounting sometimes to complete melancholy; more rarely maniacal attacks, mental derangement, aphasia, sleepiness.

Diminution of mental activity.

Amblyopia and amaurosis, with the appearance of choked disc and neuroretinitis; inequality of the pupils; strabismus.

Violent headache, often accurately localized.

Monolateral anæsthesia, usually in the form of anæsthesia dolorosa; neuralgia.

Monolateral paralysis of varying degree, gradually increasing; jerking; quivering; cramps of the affected groups of muscles, developing sometimes into epileptoid attacks.

3. *Terminal symptoms.*

Imbecility; total want of energy; sopor; and comatose conditions.
Gradual extinguishing of mental activity.
Widespread anæsthesia.
Very hesitating speech; paraplegia.
Incontinentia urinæ et alvi, or else retention.
Increased temperature of the body.
Symptoms of meningitis; apoplexy.

B.—Symptoms according to the Nature of the Neoplasm.

It is here sometimes difficult to draw the dividing line, inasmuch as the neoplasms do not always maintain a continual, and still less a constantly typical growth. A slowly growing glioma may become rich in cells, and then increase rapidly in size; and a tubercular tumor, which at first shot up rapidly, may undergo retrogressive changes, and become calcified. The following observations, therefore, which suggest some variations in the symptoms and course for the various kinds of neoplasm, are intended only as an attempt to establish a probable diagnosis.

The following speak for **Glioma:**

Commencement of the symptoms being preceded by a considerable injury of the skull.
Slow progress of the symptoms, and hence relatively long duration of the illness.

Isolated occurrence of the tumor.
Intercurrent apoplexies.
Good state of nutrition of the patient.

The following speak for **Tubercular Tumor:**

Hereditary predisposition to tuberculosis.
Occurrence in childhood.
Seat in the cerebellum.
Tuberculosis of other organs.
Commencement of the symptoms, after acute febrile diseases—for instance, measles.
Intercurrent febrile paroxysms (?), with increase of the cerebral symptoms.
Multiple occurrence.
Complication with meningitis.
Absence of apoplexies.

The following speak for **Carcinoma:**

Rapid progress of the symptoms.
Solitary (cerebral) occurrence of the neoplasm.
Perforation of the bones of the head.
Carcinoma (or melanosarcoma) in other organs.

Proof of *infection* or *invasion*, furnished by the history and by other manifestations, speak for *gummata* or *echinococci;* these subjects, however, have been treated in another volume of the Cyclopædia.

C.—Symptoms according to the Situation.

We would here recommend all possible caution, and would remind the reader of what we have already said about the dependence of the symptoms of tumor upon so very many conditions, which even at the autopsy remain unexplained. In the first place the nature and extent of the symptoms will depend more upon the mode in which the tumor affects surrounding parts than upon any other one thing. How can one with propriety compare slowly growing neoplasms, which scarcely injure nerve paths, lying in actual contact with them, with such as destroy entire territories of the central nervous system in their rapid march? And of what use are the most extensive statistics, when both quickly growing and retrogressive tumors of the same locality are classed as equivalents in respect of their symptoms? If, then, in spite of this, we collect together, below, the symptoms of tumors according to their position, we shall endeavor to employ the most similar cases, and, before all, not to differentiate too minutely. It will of course be understood that the general symptoms of tumors are to be added to those set forth under the different headings.

1. *Tumors situated close to the convexity.*

As here a direct advance of the tumors towards the meninges takes place, they are usually attended by considerable headache, which may be sometimes precisely localized. Paralyses and disturbances of sensibility are not generally well marked, widespread, or persistent, while epileptoid attacks are more frequent.

Frontal Lobes: psychical disturbances, occasionally passing over to the condition of exaltation.

Frontal headache, disturbances of the sense of smell, scarcely any disturbances of sensation or motion. If the neoplasm extends over to the second or third frontal convolution of the left side, aphasia makes its appearance.

Case I.

I. P., aged forty-nine, from Bonn, is said to have suffered from vertigo and cramps in early youth, but subsequently to have become healthy and well developed. When twenty-one years of age, however, another attack of vertigo is said to have occurred. Since 1843 the patient has been married, and the following particulars were communicated by his wife. She says that her husband has always been excessively stupid, allowing himself to be made a fool of in all directions—a statement which I have had confirmed from other sources. Ten years ago a violent bleeding from the mouth and nose took place, in consequence of which a very bad-smelling nasal discharge was developed. Frontal headache, especially of the left side, and attacks of giddiness, exhibited themselves now and then, while still the patient carried on his daily labor with his usual diligence. In October, 1864, a small tumor, the size of a small pea, appeared in the inner and upper angle of the left orbit. It was painful on pressure, and became gradually larger. The eye became pushed more and more outwards. In February, 1865, the patient suddenly ceased working, with the observation that he could work no longer, for everything seemed confused before his eyes. He remained constantly in his room, and could not be induced to undertake even the lightest occupation. Shortly after he had recovered from a three weeks' attack of jaundice, repeated bleedings from the nose occurred in the middle of March. The patient began to perform all sorts of absurdities, loosened the panes of glass out of the window-frames, remained often for hours standing on the same spot, and spoke little. Frontal headache, particularly towards the left, was constant during this time. In June the patient often lit a candle in broad daylight, "because it was too dark in the room." The appetite and other functions were regular, the bowels alone being somewhat constipated. Early in August I was requested to see the patient for the purpose of giving an opinion concerning his mental state.

Status præsens.—The patient is of middle stature, sallow complexion, with rather pale mucous membranes; panniculus adiposus moderately developed; muscular development strong; skeleton powerful; head small, dolichocephalous; forehead very narrow, low, and retreating. In the inner upper angle of the orbit, a tumor, of the size of a large hazelnut, is situated. It has an uneven surface, is moderately hard, is painful on pressure, and is not movable upon the inner wall of the orbit from which it grows. The skin covering it is but slightly movable. The left eye is dislocated considerably outwards; the right eye also deviates outwards, without any mechanical reason being apparent in the orbit. The pupils are rather dilated, and react sluggishly and imperfectly; a difference between them cannot be noticed. The acuteness of vision is greatly diminished—the patient not being able to count fingers at a distance of two feet. While this test is being made, the patient becomes stupid and confused, and further examination must be desisted from. It can only be added that the simple inspection of the eyes does not detect any great alterations in the refracting media. The mouth is but slightly drawn down to one side; tongue straight; speech slow but not hesitating; lungs and heart without any anomalies worthy of note; belly somewhat drawn in; bowels rather confined; muscular action slow but powerful; gait slow. No anæsthesia can be found anywhere.

The patient speaks little; gives replies to questions slowly; and does not usually keep to the point, but clothes his answer in general remarks. Notwithstanding the consciousness of his destitute condition, a certain amount of self-esteem pervades his entire conversation, manifesting itself especially in the unboundedness of his wishes. Sometimes, too, while he sits vacantly gazing before him, he makes motions with his fingers, which are easily recognized: "he counts money." Notwithstanding the most careful investigation, I could not ascertain that any hallucinations of sight or hearing were present.

The patient suffers from continuous headache, and complains of it most in the right side of the forehead. In other respects he does not complain of painful or perverted sensations.

Upon the basis of the above, I gave it as my opinion that the patient was laboring under a disturbance of intellect, caused by a tumor which was connected with that situated in the inner and upper angle of the left orbit.

I saw the patient in the beginning of September, and again in the middle of the same month.

He spoke hardly any, remained always in bed, passed everything under him, and presented in general the picture of a complete break-up. On the 23d of September the patient is said to have had convulsions, which commenced on the left side, and gradually extended to the right. Since then he has remained in a soporous condition. On the 25th of September, towards evening, death took place.

The *post-mortem* examination was made by me on the 26th of September, twenty-two hours after death.

Cavity of the skull: Calvarium tolerably closely adherent to the dura mater; moderately thick; in the region of the frontal eminences quite sclerosed; and at the right, four and a half lines, at the left, six lines thick.

Dura mater anæmic; along the sinus longitudinalis, slight Pacchionian granulations. In the longitudinal sinus a coherent blood-clot of about the consistency of fat pork. On the inner surface of the dura mater, which has been removed at the level of the section through the skull, there appear upon the right side and anteriorly, about two inches from the middle line, abrupt tumor masses slightly adherent to the pia mater, which in the centre have a thickness of three lines, and are of a grayish-red appearance. The surface covered by these masses is triangular in shape; the base, situated at the section through the dura mater, being three and one-quarter inches long, and the height two inches. The external surface of the dura mater is quite smooth. The pia mater is only slightly injected, but extremely œdematous. At the place where the tumor masses, situated on the inner surface of the dura mater, are adherent to it, it has assumed a tendinous opacity. The brain is free of blood, tough, and contracts on the cut surface. The gray substance is but slightly developed. The left ventricle is greatly dilated, and partially filled with clear serum. Upon the right side and anteriorly the white substance is considerably discolored. The right lateral ventricle is not so dilated as the left. The anterior cornu is wanting; but, instead of it, the ventricle is divided transversely by a highly vascular septum, which proves to be an inversion of the ependyma. This inversion is produced by a very vascular, irregular tumor situated upon the surface of the right anterior lobe, which is distinguished from the brain substance by its greater hardness, and is immediately surrounded by a zone of grayish gelatinous tissue. In order to make out accurately the relation between this tumor and that in the orbit, the scalp was now dissected off as far down as the commissures of the eyelids in front, and behind down to the neck, and a section made almost horizontally with the saw from the root of the nose towards the ephippium. By the aid of two other lateral vertical sections, the preparation was obtained which I exhibited in the Lower Rhenish Physiological and Medical Society. Before I describe it, a few words on the parts left below that horizontal section. The perforated plate of the ethmoid is completely destroyed. In its place there lies a tumor about the size of a large hazelnut, the cut surface of which presents a marrow-like appearance; a part of this tumor also extends into the left superior nasal meatus.

The nasal septum has been pushed greatly to the right by the tumor. The lamina externa of the perforated plate has been pierced on both sides by tumor masses, which on the right have formed a narrow tumor along the posterior half of the inner wall of the orbit. On the left the walnut-sized tumor situated along the inner wall of the orbit has been partially removed with the preparation. The eye has been displaced considerably outwards by it. The optic nerve follows an arched course around the tumor.

Our preparation exhibits the following conditions of the parts: A tumor, the size of a large walnut, is situated slightly to the right of the middle line, external to the dura mater, at a spot corresponding to that where the right olfactory bulb always lies. This tumor is intimately adherent to the dura mater over a surface which would be covered by a shilling. The new-growth has a mesh-like appearance, is of a grayish-red color, and rather soft. Between this tumor and the bone there is a quantity of creamy pus. The vitreous table of the frontal bone, and the crista galli of the ethmoid, are completely destroyed, so that the frontal sinuses are opened over a space two inches in breadth, by one and a quarter inches in height. The bone, which has been absorbed in places to a depth of one and a half lines, is partly rough, and partly covered with connective tissue. On the inner side of the dura mater, separated from the above tumor by some very fine tendinous remains of this membrane, is found the previously mentioned tumor, with uneven and highly vascular surface, and of tolerable consistence. It is three inches long, two and three quarters inches broad, and two and a quarter inches high, and fills the right anterior fossa and a large portion of the left, so that the falx cerebri and the left anterior lobe of the brain are considerably displaced outwards and backwards. The surface of the tumor has a reticulated appearance, and upon the section it has a marrow-like appearance. Here and there in its centre there are yellowish spots. Upon the inner surface of the dura mater, in the neighborhood of the tumor, masses of the new-growth are deposited four and a half lines deep, which sprout up at intervals, and present those abrupt elevations already described. The dura mater is smooth on its outer surface; where it covers the tumor, it has the appearance of an external layer of the growth, and can be distinguished from the latter by the white margin which it presents upon a section; it may even be separated from the tumor with ease.

—*Obernier*, Virchow's Archiv. Vol. XXXVI. p. 155.

Parietal lobe: Slight hemiplegia and unilateral disturbances of sensation, which are the more pronounced the deeper into the brain the neoplasm penetrates. Disturbances of motion in certain groups of muscles are to be looked for. We have referred to these in the sense of the experiments of Fritsch, Hitzig, Nothnagel, and Ferrier. When the tumor extends towards the region of the left island of Reil, aphasic disturbances may be expected. (See the drawing on page 240, and the case briefly detailed there.)

Posterior lobe: Diffused headache; except the disturbance of vision, deterioration of the senses is rare; no considerable disturbance of motion; vertigo and convulsions common. (Nothnagel's convulsive centre in rabbits.)

2. *Tumors of the base in the neighborhood of the optic chiasma.*

The disturbances which the chiasma and optic tract suffer, as well as the other nerves springing from the base of the brain, serve here as guides for the diagnosis.

Anterior to the chiasma nerv. optic.: Disturbances of the sense of smell; injury of the fibres of the optic nerve, which pass to the inner half of the retina, and consequently hemiopia, in which the defect lies to the outside in the field of vision of each eye.

Symptoms of tumor of the frontal lobe.

Case II.

About a year ago, P. S., from Godesberg, presented himself in my out-patient form, on account of a diminution of his sight, which had come on a few days previously. The examination showed that the acuteness of vision in the right eye $=\frac{1}{8}$, and in the left $=\frac{1}{6}$, the excentric parts of the field being normal. A slight hypermetropia was the only thing abnormal which I could find with the ophthalmoscope, nor did the most careful investigation detect any disease in other parts. The nature and cause of the disturbance of sight remained at first quite unknown to me, and I kept the patient under observation upon neutral treatment. In four days the vision was reduced to one-twentieth without any ophthalmoscopic change being visible. At the same time disturbances took place in the previously unimpaired general health. The patient complained of slight headache; he lost his appetite; his sleep was restless; and his pulse became somewhat rapid. From the negative ophthalmoscopic appearances, the cause of the disturbance of vision was evidently extra-ocular, and, from the simultaneous occurrence of it in both eyes, probably also extra-orbital; while the absence of those symptoms which indicate a material disease at the base of the brain, pointed to a localization of the cause at the basis cranii. Nothing, however, could be discovered concerning the nature of the disease; the anamnesis let in no light on the question. I ordered the patient to be cupped on the temples, and prescribed calomel internally. In five days the acuteness of vision in both eyes was reduced to one-half, although the fields of vision remained of normal dimensions. There was no increase in the disturbances of the general health; there was no fever; and the strength of the patient was such that he could be treated as an out-patient. I now applied the artificial leech several times, but could only observe that the vision sank rapidly to quantitative perception of light, until, finally, in the course of three weeks (from his first visit), it was completely extinguished. He remained in this sad state for nineteen days. On the twentieth day return of sight to the right eye could be ascertained by aid of concentrated light, and two days afterwards sight began to return to the left eye in the same way. With the return of the perception of light the general symptoms disappeared, only that the patient was still easily tired and was not quite free from headache. In the course of the next few days the vision increased so much that an examination of the excentric fields could be undertaken. It showed a want of the external halves of the fields of vision.

The transition of the existing portions of the fields of vision to the lost portions was effected by a region, which, by a low light, should be reckoned to the latter, so that then the boundary-line of the defect fell somewhat to the outside of the fixation-point—running in the right eye in a vertical direction, and in the left diagonally from the inside and above downwards and outwards. Within the next four weeks the central vision increased in the right to $V = \frac{1}{2}$, and in the left to $V = \frac{1}{20}$, while the defect in the excentric vision continued in the way described.

In the spring and summer, during which period I saw the patient, on the average, once in three weeks, no alterations occurred either in respect of the acuteness of vision or of the general health. He was able again to perform light household duties. In August he suffered from typhoid fever, with which his mother and sister were attacked at the same time. This illness did not influence the previous condition, as subsequent observation proved. Even on the 25th of October I obtained no other than the above-mentioned results, either by an investigation of the functional derangements or by the ophthalmoscope. On the 30th of the same month I was informed of the death of the patient, which had taken place ten hours previously. As I was informed by Drs. Schwann and Finklenburg, the patient had been attacked on the 28th with symptoms of acute meningitis, which had ended fatally during the night of the 29th. At the autopsy next day we found, in the first place, signs of purulent meningitis, and, further, the suspected tumor.

It was about the size of a pigeon's egg, and lay between the trunci optici, and in front of the chiasma, and was surrounded by the optic nerve in a forked manner, the nerve fibres being parted by it. Upon a section of it, an ichorous, bloody fluid poured out. Besides this tumor, however, there was a second, situated beneath the pons, unconnected with the first, and larger than it. Both tumors, along with the optic nerve adherent to the first, were sent for examination to Prof. O. Weber. He says: "Both tumors were sarcomata of a peculiar formation. The larger, more posteriorly situated, and evidently oldest tumor, had raised the dura mater, and consisted of an excessively vascular (teleangiectatic) sarcoma tissue. It had probably originated in the cavernous sinus. Peculiar capillary formations of different sizes projected from its surface. The smaller ones were solid, the larger ones vesicular, owing to cysts having been formed by the occurrence of hemorrhages into the tissue. The second tumor, which originated in the subarachnoid vascular tissue, and was situated between the optic nerves, consisted of a number of hemorrhagic cysts held together by nucleated sarcomatous tissue. Both trunci optici had undergone fatty degeneration, and exhibited here and there proliferation of nuclei in the neurilemma."

—*Saemisch*, klinische Monatsblätter, 1865, p. 51.

Laterally from the chiasma nerv. optic.—When the tumor advances towards the chiasma, disturbances of function of the optic fibrillæ; those on the side of the tumor supplying the external half, those on the opposite side supplying the inner half of the retina; hence right or left-sided hemiopia.

Neuralgia and anæsthesia of the fifth nerve (generally in all its branches); paralysis of the motor portion on the side on which the tumor is situated.

Diminution of sensation and power of motion upon the opposite side of the body from the tumor. (Vide case under the heading Pons.)

Behind the chiasma nerv. optic.—Lesion of the optic fibrillæ which pass to the outer half of the retina; hence hemiopia, with the defect inwards on each side.

Disturbances of motion of the eyeballs.

Unilateral paralyses.

The so-called circus-movements.

3. *Tumors in the corpus striatum and nucleus lenticularis.*

In respect of the paralytic symptoms, they afford the picture of an ordinary apoplexy; only they appear in association with other tumor symptoms (*e. g.*, alterations in the optic nerve and retina), develop gradually—not suddenly—and are attended by symptoms of irritation.

4. *Tumors of the cerebellum.*

Occipital neuralgia; no disturbances of sensation; vertigo; disturbances of the power of co-ordination; unsteady gait; forced motions backwards; disturbances of the motions of the eyeballs, and disturbances of vision; occasionally disturbances of the sensation of hearing.

Case III.

The patient, aged forty-three, was a tailor. On the 8th of November, 1866, he was admitted to the Charité Hospital, under the care of Prof. Griesinger, where he remained until his death, about six months afterwards (15th of May, 1867). Since Christmas, 1865, he had suffered night and day from very violent headache, and also for the last three months from vomiting in the morning. These symptoms, however, had become less troublesome of late.

Since last winter an increasing amblyopia, ushered in with subjective appearances of light and mouches volantes, had made its appearance; and this obliged him, in July, 1866, to give up his employment, and at the time of his admission had already advanced to complete blindness. The patient, nevertheless, believed that he saw, although indistinctly; but this was a mere hallucination. For the last fortnight the legs had become somewhat weaker.

The examination of the eyes, made (Prof. von Graefe) on the 27th of November, showed: "Double optic neuritis, of the form which is found with increased intracranial pressure. This is already retrogressive, and is better marked in the left than in the right eye. It probably does not extend up the nerve. Complete amaurosis in each eye. Along with this, a restriction in the mobility of the eyes seems to exist, particularly in the direction of the interni, more so of the left internus; both pupils very dilated and motionless, the left one somewhat the wider of the two." Among the other symptoms, a disturbance of motion of the lower extremities was especially to be noted. The patient's gait was unsteady; he diverged always from the straight direction in walking, usually towards the left side; sometimes he ran round in a spiral towards the left. At a later period these tendencies increased to such a degree that occasionally the patient could not even stand alone. For a length of time a well-marked, involuntary, retrograde motion took place whenever he attempted to stand or to walk forwards. Slight left facial paralysis; retention of urine; sexual impotency; sensation intact. Almost complete deafness of the right ear existed; but this seemed to be of old standing. (Dr. Lucæ found a milky turbidity of the membrana tympani of the right ear.)

The patient continued to have headache on and off, particularly in the occipital region, and percussion of the skull at this place, especially towards the right side, was painful. Vomiting rarely occurred. The stupidity of the patient increased, and soon after his admission he was liable to hallucinations. A peculiar way of holding the head gradually became developed; it was bent quite backwards, and rotated to the left. The face was also turned slightly to the left.

An ophthalmoscopic examination, made on the 16th of April, 1867, gave very much the same results as the former one. The somewhat more minute description dictated by Prof. von Graefe at this time ran as follows: "In both eyes a very well-marked choked disc, in the stage of transition to white atrophy of the optic nerves. The elevation is not now steep, but still continues distinct. The smaller arterial vessels are disappearing, while the large veins continue to be overfilled and tortuous. The tissue of the optic disc is chiefly white and opaque; towards the margins only there are some remains of a reddish-gray infiltration, indicating the presence of apoplexies." About four weeks afterwards (11th of May) I found the white opacity around the optic discs much diminished in each eye, so that it extended but slightly beyond the margins of the discs. The prominence of the discs had also become reduced. In the last few weeks of the patient's life a ptosis of the left eye was present. A more accurate investigation of the motions of the eyes was not, however, possible, owing to the extreme imbecility of the patient.

The autopsy (Dr. Cohnheim) showed, as had been diagnosed, a tumor of the cerebellum on the right side, besides miliary tuberculosis of the lungs, tuberculous pyopneumothorax of the right side, and chronic hypertrophy of the spleen.

A considerably increased pressure had existed within the cranial cavity. The dura mater and pia mater were tightly stretched, and the sulci in both hemispheres almost quite obliterated. The dura mater was of normal thickness, and pale upon its inner surface; the pia mater, on the other hand, was dull and opaque, and even tendinous along the course of the larger vessels. The cerebrum was removed, so that the tentorium and anterior part of the pons lay exposed. It could now be seen that the right half of the tentorium bulged forwards more than the left, and that the pons, as also the superior vermiform process, was pushed to the left. After removal of the tentorium, a tumor mass was seen protruding between the right margin of the cerebellum and the posterior margin of the petrous portion of the temporal bone. This tumor was solid anteriorly, while posteriorly it consisted of a fluctuating, yellowish, transparent cyst. The mass extends half an inch beyond the internal auditory meatus in front, and posteriorly to the point where the horizontal part of the transverse sinus unites with the bent part. In the neighborhood of the internal auditory meatus, the tumor is closely adherent to the temporal bone, and, when it was separated from it, two deep, circular depressions were found in the substance of the bone. In the remainder of its extent, the surface of the tumor is connected with the inner surface of the dura mater by a number of thread-like adhesions, which contain vessels, and may readily be broken down. After removal of the cerebellum, the greatest length of the tumor is found to be, from before and the inside to behind and the outer side, two and a half inches, of which one and a half inches appertain to the solid part of the growth. Its broadest dimension lies in the direction from within outwards, and amounts to two inches in the solid part, and to one and a quarter inches in the cystic part. The greatest height in the solid part is one and a half inches, and in the cystic part one inch. The entire tumor is covered by a process of the pia mater, which may be separated from it in many places without difficulty. The solid portion of the tumor is uneven and lobulated. It shines through the pia mater with a grayish-white color in some places, a yellowish-white in others; here and there it presents even a gelatinous appearance, and again in others it seems hemorrhagic. The cystic portion is non-vascular, and presents a transparent, yellowish-green appearance. Upon a section the tumor is found to be not very firm, and rather elastic. The surface of the section, especially towards its periphery, displays numerous transparent, gelatinous spots. Towards its inner side the cut surface becomes yellowish, and in its lower part numerous sections of distended vessels and many punctated hemorrhages may be seen. The cystic portion is not quite so large as a walnut, and is easily separated from the remainder of the tumor, with which it seems to be closely adherent throughout only a small extent. Still more accurate examination confirms the idea of the growth being a myxo-sarcoma. Towards the inner side it may be separated, to a great extent, without trouble, from the cerebellum and the crura cerebri; in the neighborhood of the third superior lobe (*des dritten obern Lappen*) alone is it intimately connected with the cerebral substance. The right anterior peduncle of the cerebellum is very much stretched, flattened, and thinned by it. The fifth nerve is flattened between the tumor and the pons, but still continues quite white. The external parts of the three anterior, superior, and inferior lobes of the right cerebellar hemisphere are everywhere atrophied; in some spots the gray matter is quite absent; in others it is very much diminished. The neighboring parts of the white matter of the cerebellum exhibit no alterations on a section.

The cerebrum is anæmic. Both lateral ventricles are greatly dilated, and filled with a clear fluid.

Both optic nerves are white, and tolerably round. While still fresh, the retinal vessels in the right eye are imperfectly visible. The optic disc seems rather broad, swollen, and somewhat prominent, and of a dull gray color.

—*Leber*, Graefe's Archiv. Vol. XIV. Part II. p. 363.

5. *Tumors of the corpora quadrigemina.*

Excessive disturbances of the motions of the eyes on both sides (Adamük); very considerable disturbances of vision; and more or less well-marked unilateral paralyses.

6. *Tumors of the pons.*

Neuralgia, anæsthesia, and paralysis of the fifth pair. Disturbances in the functions of the fourth and third nerves, when the growth tends forward; when it tends backward, paralysis of the sixth and portio dura of the seventh. These paralyses are situated generally upon the opposite side from those in the extremities. Difficulty of swallowing; disturbances of function in the urinary bladder; rarely convulsions.

Case IV.

According to the report of the physician under whose care the patient was, Johann V., a porter, aged forty-one, became affected, in the spring of 1858, with double vision, and violent pains in the back of the head and neck, which latter increased when movements were made. Later on, very severe neuralgia of the supra- and infra-orbital divisions of the trifacial made its appearance. When this had existed for some weeks, opacities of the right cornea became observable, which resulted in blindness and atrophy of the globe. Loss of sensation now came on in the whole of the right side of the face and mouth, and mastication became difficult on the same side. In September, within a short period, paralysis of the left side of the face and body was developed, and twitchings and a sensation of formication were occasionally felt in the paralyzed parts. Incontinence of urine came on gradually; the bowels were moved with difficulty; and now and then attacks of palpitation of the heart, attended with difficulty of respiration, were complained of. The following condition was noted upon the patient's admission to the hospital on the 19th of November: His mental faculties are intact; his speech somewhat rapid. The right eyeball is sunken; the cornea and iris opaque and pushed forward; the vision completely extinguished; sensation in the conjunctiva wanting; motion of the globe outward much restricted. The right side of the face, and the entire mucous membrane of the mouth, are without sensation; while motion in these parts is moderately good. The left side of the face droops. During motion, such as speaking, crying, and laughing, the face is drawn to the right. When protruded, the tongue inclines to the left. The left arm is moved with greater difficulty than the right; and the left leg is completely paralyzed, and its sensation reduced. Pressure on the back of the neck, close to the occiput, gives some pain, and there is occasional spontaneous pain in the occipital region itself. Bitter substances cannot be tasted upon the right side of the tongue. The functions of the right ear are normal. The urine is alkaline, and is passed involuntarily. The bowels are excessively constipated. Pulse 76, and small; respiration regular; no abnormal symptoms of other organs. A considerable bed-sore exists in the region of the sacrum.

Until his death, on the 14th of December, the patient complained much of pain in the right side of the face and in the region of the bed-sore. The appetite became impaired, the sleep broken, and he suffered sometimes from violent pain in the left leg. The bed-sore spread from the sacral region towards the trochanters. The general feebleness increased. The pulse became rapid and small. Consciousness was retained to the last. Attacks of difficulty of breathing, or of palpitation of the heart, were not observed. Gradual collapse came on, and death occurred without convulsions.

Autopsy.—Twenty-two hours after death.

Calvarium normal. The dura mater here and there slightly opaque; its tension moderate, and its vascular injection normal. Coagulated blood in the longitudinal sinus. The arachnoid somewhat œdematous. The pia mater containing a moderate supply of blood. At the basis cranii, to the right of the sella turcica, where the fifth pair passes through a fold of the dura mater to reach the Gasserian ganglion, a neoplasm is situated. It is yellowish in color, difficult to incise, and resembles pork somewhat in appearance. It is about three lines in height and one inch broad. The whole of the fifth nerve seems completely embedded in it. On the remaining surface of the skull there is nothing abnormal to be seen. At the base of the brain, on the right half of the pons, two yellowish bodies, each of the size of a cherry-stone, and of a tough consistency, are seated. One of them lies exactly at the point of exit of the fifth pair, which disappears in the tumor. The other neoplasm lies free of nerves. The tumors have a firm, pork-like, dry appearance, and extend six lines deep into the substance of the pons, which, however, even in th immediate neighborhood of the tumors, retains its normal consistence and colo The other parts of the brain display a normal condition, except the ventricle where a slight increase of serous fluid is observable.

The examination of the other organs reveals no pathological appearances, wit the exception of the lower lobe of the left lung, which, at its base, displays a dee drawing-in, with thickening of the pleura, upon a section of which, firm, grayis cicatricial tissue is seen, in which some dilated bronchi run.

—*Ruehle*, Greifswalder Beiträge, Vol. II. 1864, p. 62.

7. *Tumors of the medulla oblongata.*

These produce disturbances of sensation, convulsions, and more rarely, paralysis of one or more of the regions supplied b a great variety of nerves; and where paralysis is present, it ma rapidly become very extended.

Difficulty of speech and of deglutition, and paralysis of th bladder are more common symptoms.

Sugar is sometimes found in the urine.

The picture is often difficult to distinguish from that c glosso-pharyngo-labial paralysis.

Case V.

Gustav Strobel, aged twenty-four, says, that until five years ago, with the excep tion of scabies, from which he suffered from his fourteenth year on, he had alwa been healthy, but had had a great tendency to perspire, and had habitual perspir tions of the feet. In the year 1858, without any assignable cause, attacks of vertig made their appearance. These recurred at intervals of various length, sometim several times during the day. They were of different length and differe severity. The lighter attacks—in which the gait of the patient was unsteady an staggering, so that he was often believed to be intoxicated—continued often f several hours. Severer attacks, in which the patient was unable to remain stan ing, but fell to the ground, occurred much more rarely, and passed off in a fe minutes. During the attacks the patient never lost his speech or consciousness, n were the attacks ever attended with spasmodic contractions. About the same tim periodic vomiting appeared, both in the morning with an empty stomach, and aft meals. Strobel distinctly asserts that this vomiting was not attended by any dy peptic symptoms, that he enjoyed a good appetite, and had no sensation of pa or pressure in the region of the stomach. At that time the patient suffered fro frequently recurring pains in the back of the head and neck. He cannot sa whether these pains and the severer attacks of vertigo coincided with the attac of vomiting, or preceded them. It also cannot be ascertained what immediat causes give rise to or aggravate the vertigo, headache, or vomiting. All the remaining functions seem to be in order, and the bodily activity and nutrition, and the appearance of the patient, leave nothing to be wished for. With the use of a neutral treatment (drinking acorn coffee), the vomiting and the pains in the back of the head gradually disappeared in the course of half a year; but the attacks of vertigo continued, and for a length of time they formed the only symptom from which the patient complained.

In the spring of 1860 Strobel was called out for military service as perfectly healthy, notwithstanding his protestations to the contrary. During his time of service, which lasted until the autumn of 1861, violent attacks of vertigo frequently occurred, especially during drill, so that he fell to the ground. Several times after such attacks he was brought to the hospital; but was generally discharged again in a few days as a malingerer, owing to his healthy appearance. Strobel says that he lost his pedal perspiration during his military service, from a severe wetting. In the autumn of 1861 the patient was discharged, and began work with a builder in Heilfingen. For the first six or eight weeks he felt quite well, with the exception of occasionally recurring violent pains in the occipital region, and radiating from that place towards the back and lumbar region and lower extremities. The attacks of vertigo became more frequent and more severe, and obliged the patient to give up his situation in Heilfingen. A physician, whom he consulted at that time, prescribed local bloodlettings on the head; but these were without benefit. In December, 1861, and in January, 1862, Strobel presented himself in the out-patient department of the Clinique several times. At this period the attacks of giddiness were so violent and continuous, that the patient was obliged to give up even the light field labors which he had hitherto performed. He could only walk with the aid of a stick, or he supported himself by large objects at hand. Repeated applications of sinapisms to the feet, in order to restore pedal perspiration, were employed for a length of time without benefit. The patient said that the violent pains in the occipital region were most severe before the attacks of vertigo; he also complained of such violent tinnitus that it often seemed to him as if some one were shouting into his ears. The patient also mentioned that he had erections remarkably often, and that semen was mixed with the urine he passed. No paralysis, disturbances of the special senses, nor impairment of the mental functions were apparent. In the spring of 1862, pressing and pulsating pains in the upper part of the head were added to the pains in the neck. It seemed to the patient as if some one had hit him "with a log of wood" on the head. The radiating pains in the feet had disappeared, and an alternating sensation of heat and cold had taken their place. In the course of the winter disturbances of vision were added to these symptoms. The patient saw everything through a fog and indistinctly. His relatives, moreover, remarked that Strobel hesitated in his speech, and that it was becoming always more difficult to understand. They also remarked that the act of swallowing was not normally performed. In eating, the morsels often went the wrong way, so that violent coughing was brought on, and the food returned through the nose. The passing of the urine, finally, had become difficult, so that the patient could empty his bladder while in the standing posture only, and with great pressure.

His relatives had observed, moreover, an alteration in the patient's manner. They said that it had become somewhat childish, and that he was easily vexed. Still he sometimes spoke in a perfectly sensible way, and, when excited, could be quieted without difficulty.

Status præsens, 21st of May, 1863.—Strobel is a large-boned man, of middle stature. The formation of the skull displays nothing abnormal. The visible mucous membranes are neither very red nor very pale. The adipose tissue is weakly developed, while the musculature is strong.

The patient complains most of violent pain in the head, situated not only in the occipital region, but also spreading out over the crown of the head, and into the upper jaw, and accompanied with a sensation as if the teeth were loose. In turning the head and in bending it backward, the pains are very much increased, and he then also experiences " noises in the head." Sometimes the pain extends to the eyes. On pressure upon the two upper cervical vertebræ, the patient draws a wry face, and complains that the pressure gives him pain. He suffers still more if the carotids be pressed back against the transverse processes of the upper cervical vertebræ. During this proceeding he complains not only of pain, but also that he "can't breathe;" he closes the eyes, and makes noisy, long-drawn inspirations, so long as the pressure is continued. The patient is hardly ever free from vertigo. From time to time the vertigo increases to violent attacks, in which he tumbles backward, and falls to the ground.

The patient wears an apathetic expression of countenance; the left angle of the mouth stands rather lower than the right; the left nostril is somewhat narrower than the right. When he laughs, the naso-labial folds on the left side are deeper than on the right.

In frowning there is no asymmetry of the wrinkles on the forehead. The muscles innervated by the fifth pair contract equally on both sides upon the application of a weak induced current by means of damp electrodes. Both sides of the face, when tested with metallic electrodes, exhibit normal sensibility.

With regard to the organs of the special senses, the sense of smell is intact. The taste is very much blunted. A few grains of picro-nitrate of potash, placed upon the tongue, do not give the patient any annoyance. Only after some time, and upon repeated inquiries, he says that it tastes bitter; while a much smaller quantity, placed upon the tongue in other people, produces a most disagreeable and highly bitter taste, which continues for a long time and obliges them to spit out repeatedly. Sour and salty matters he often confuses; on the other hand, he recognizes sweet substances with comparative ease.

With the right ear the patient hears well, distinguishing the tick of his watch at a distance of three feet. But with the left ear he hears so badly that he can only perceive the watch at a distance of one inch. The continuous noises in the ears, formerly so troublesome, have reappeared of late, now and then.

An investigation of the power of vision, and of the objective changes in the eyes, was undertaken by Drs. Klein and Teuffel, with the following result: The eyelids are in constant winking motion. The superficial conjunctival vessels exhibit a considerable amount of injection in the left eye, particularly in its lower half. The injection becomes gradually closer towards the margin of the cornea, which latter, over a space, in its lower fourth, about a millimetre broad, is of an opaque gray color. The limbus conjunctivæ is not swollen. In both eyes there is a moderate amount of exophthalmus, rather more marked in the left than in the right (the patient's relatives had for a length of time been aware of greater prominence and redness of the left eye). The motions of the left globe were restricted in every direction. The left globe also stands somewhat higher than the right. The patient is short-sighted in the left eye, and his short sight cannot be completely corrected by concave glasses. There is paralysis of accommodation in the left eye. Ophthalmoscopic examination: The optic disc enlarged, not distinctly bounded, darker and more opaque than normal; the vessels within its margins are indistinct; outside of them the arteries are empty, the veins broad and overfilled. Diagnosis: Commencing optic neuritis due to venous congestion.

The motions of the tongue are effected normally, although slowly. When pushed out, its point deviates somewhat obliquely to the left. The uvula does not hang more to the one side than to the other. The left arch of the soft palate hangs rather lower than the right. The patient's articulation is very much impaired, so that his speech can be understood with difficulty, particularly when he is excited. The speech is thick, similar to that of an intoxicated person. It has been ascertained that he produces certain words and letters with greater difficulty than others.

The motions of swallowing have become very difficult. The patient stops after each mouthful of water. When he takes his soup, unless he is very cautious, a part of what has been taken into the mouth returns through the nose. He is quite unable to swallow dry bread, but first dips it into water. An intelligent patient, occupying the same ward, says: "It seems as if he wanted the proper pressure in his throat."

The position of the body is almost always bent forward, with the hands resting upon the knees; sometimes he sinks with his head and the upper half of his body still more forward, until he raises himself up again. It has even attracted the attention of the other patients how little power Strobel has in his back, and how easily he falls together. They say: "He is quite strong in the legs; it seems only as if he were paralyzed from the small of the back upward." And, in fact, the muscles of the upper and lower extremities on both sides can be contracted with normal power; at most the pressure of the right hand is a shade weaker than that of the left. As long as he is lying down, the most complicated motions of the extremities can be performed without any difficulty, and his muscular sense is perfectly normal; but the patient shakes and staggers as soon as he is placed upright.

He is able to accomplish only a few tottering steps without support. He makes use of a stick, or prefers to support himself by the objects of furniture in the room, when he wishes to go from one place to another. When he goes from one bed to another, he can do it with greater ease if he goes quickly than if he takes time.

The sensation of touch and pressure in the extremities is normal. The pains in the lower extremities—of which the patient complained so much during the previous winter—have disappeared, and have given place to a sensation of "buzzing," of numbness, and, above all, of cold. His fellow-patients say that Strobel generally seeks the places in the room where the sun shines in, and stretches his feet out in its rays. The temperature between the first and second toes is 21.3° Centigrade (normal, 33.9°); while the temperature in the closed hand is 35.7° (normal, 36.1°). The patient has often of late complained of shooting pains in the upper extremities, and a feeling of numbness there.

The bowels are constipated, being often not moved for several days, and even after purgatives only every second or third day. The vomiting, which formerly accompanied the attacks of vertigo, has not reappeared of late.

The urine is secreted abundantly; specific gravity, 1016; reaction acid. The urine deposits no sediment, and contains neither albumen, sugar, nor spermatozoa. The patient says that he experiences a shivering when he passes water; that the water at first goes back; that he must press greatly; and that he can urinate in the erect position alone. The previously very frequently recurring sexual excitations have become rarer of late. Erections occur in the morning only, now, and the discharge of semen with the urine has ceased, according to Strobel's account. His linen shows no trace of spermatozoa.

The patient's memory has suffered no great impairment so far, and his replies are sensible; but the psychical processes seem to be performed slowly and with diminished energy. He is generally in a quiet humor, but quickly changeable; and the entire manner of the patient conveys the impression of his being silly. During his sojourn in the Clinique, which continued until the end of July, no important change occurred in the condition of the patient. On some days he felt very well; on others he complained that the headache, vertigo, noises in the ears, and sensation of cold in the lower extremities, were very severe. The treatment consisted in drastic pills—without which a motion of the bowels never took place. Leeches were applied, behind the ears and on the temples, several times, without giving the patient any relief, but without aggravating his condition. We have no account of the case from the end of June, 1863, to the end of June, 1864, during which time the patient lived with his parents at Rottenburg, and was treated by several physicians. On the 29th of June, 1864, Dr. Gussmann, at that time a clinical clerk in the Medical Clinique, visited the patient at his dwelling, and noted the following status præsens: Strobel sits at the window, quite packed in with cushions, his head and body bent forwards. If he is called on to sit upright, he complies with great effort, and then sinks back again into his former position. Even while his face is at rest, one may remark that the left side hangs down loosely, that the left naso-labial fold is obliterated, that the left angle of the mouth stands lower, while the right ala nasi and the right angle of the mouth are drawn somewhat upwards. In whistling and laughing the difference between the two sides of the face becomes still more marked. In frowning there is no distinct difference between the two sides of the forehead. Both upper lids hang rather low; the orbiculares palpebrarum act equally on each side; in the coarser movements of the orbital muscles no abnormality can be found; the pupils are of medium size, equal, and react but sluggishly to light. The patient counts fingers at a distance of some feet, but cannot recognize finer objects, and complains of frequent twinkling and twitching in the eyes. The sensibility over the whole of the integument of the head and face seems normal, or at most but slightly blunted. The hearing of the left ear is so bad that the patient perceives the ticking of a watch when it is brought quite close to the ear only, while with the right ear he hears it at a considerable distance. The tongue, which is very foul in its centre, deviates somewhat to the right when protruded. The coarser movements of the tongue are performed normally, although somewhat slowly, as are also those of the soft palate. The speech is very much impeded, lisping, like that of an intoxicated person. When the patient becomes excited, his speech is hardly to be understood. Deglutition is also impeded in a high degree. Solid food passes down with less trouble; but fluids frequently return through mouth and nose, and excite violent coughing. The sensation of touch in the tongue is normal. The taste was not tested. According to the report of his relatives, the sensation of smell is retained. The arms are somewhat thin, and the hands give a remarkably cold impression. The pressure of both hands is weak; that of the right weaker than that of the left. Every motion can be performed with each arm; but those with the right are made more slowly, with less power, and less surely. The sensation in both arms is equal, and not diminished to any great extent. The muscles of the lower extremities, especially those of the calves of the legs, are much reduced in size. Flexion and extension can be effected in the hip, knee, and ankle-joints; but the motions are slow and weak. Sensation is retained in both legs, which, like the hands, give a very cold impression. If the patient be strongly supported on both sides, he can walk through the room, while at the same time his body is greatly bent forwards, and he drops his legs after him in a remarkable manner, particularly the right one. If he be left without support, he immediately falls forwards. His chief complaint is still an excessively violent headache, situated principally behind at a point corresponding to the centre of the occipital bone, and radiating from there on both sides to the eyes and forehead, and some inches down the neck. The pain is constant, but becomes often exacerbated, especially, it is said, in bad weather, by day as well as by night, and is then so violent that the patient shouts out and moans. He complains, moreover, of pains in both sides of the tongue, and on the inner surface of the right cheek, and also of a feeling of numbness in the right arm. His humor is very changeable: at one time gay, and in strong contrast with his pitiful condition, so that the patient joins in the laughter of others; and at the next moment greatly depressed, so that he begins to cry and to beg for help. By his sunken position, hanging mouth, expressionless eye, and lisping speech, the patient gives the idea of one who is deeply intoxicated; and yet his relatives have remarked no great weakening of his mental faculties, especially not any such of his memory. He himself is most anxious to be cured, so that he may enter the army again.

The patient's appetite is moderate. Vomiting never occurs. The bowels can still be moved with drastic purgatives. If the constipation continues for more than two days, the headache increases to such a degree that the entire house is set in a state of excitement, and the mother (a monthly nurse) hastens to his aid with an enema-tube. Pulse, eighty-four. The region of precordial dullness is not enlarged; no distinct impulse of the heart can be felt. Its sounds are normal. On the right side, between the scapula and the vertebral column, at a spot corresponding to the bronchus, there is bronchial breathing, but nowhere rhonchi or sibilant râles. Shallow alternates with very deep breathing. The patient is obliged to press very much, and to make four, five, or even more attempts before he can succeed in passing water. Concerning the shape of the urinal stream, he can give no information. The urine contains some red sediment. Professor Niemeyer was informed by Strobel's relations, on the 3d of September, that the patient had died with unexpected suddenness. An inquiry after his health during the past few weeks showed that Strobel, with the exception of greater weakness, and greater indistinctness of speech, had continued in the same state. Even on the morning of the day on which he died, nothing remarkable was noticed. Soon after two o'clock he was seized with a severe rigor, which continued for half an hour, and obliged him to go to bed. He then complained of severe oppression, and said that "it seemed lighter in the head, but that everything had gone to the heart." At four o'clock the patient attempted to drink some milk, but was unable to swallow, so that the milk returned through his mouth and nose. He still spoke, and could use both hands. Towards five o'clock he became unconscious, and death supervened, without any previous rattling in the throat.

The autopsy was made by Prof. Niemeyer, twenty-six hours after death, at the dwelling of the patient, under most unfavorable circumstances. It gave the following result:

The body is not emaciated; on the contrary, rather fat. The external coverings pale, but not cyanotic. On the dependent parts of the body considerable post-mortem hypostasis. Very great rigor mortis.

The skull remarkably thin, wanting in blood; the diploë almost absent. The dura mater is equally tightly stretched on both sides and pierced by abundant Pacchionian bodies. A well-defined, regular, round protuberance is formed by a group of large Pacchionian bodies lying to the left side of the sagittal suture, and a corresponding depression is found on the inner surface of the calvarium. The longitudinal sinus is empty. The convolutions are very flat; the sulci almost obliterated, so that the surface of the brain is remarkably even. In the subarachnoid spaces there is no trace of fluid. The pia mater is free from blood, and pale. The lateral ventricles, which were laid open by a very superficial horizontal section through the hemispheres, contain a large quantity (about four ounces) of a clear fluid, the posterior horns being particularly distended. Without any stretching of it, the foramen of Monro admits the tip of the little finger. The septum pellucidum is very thin and transparent. The neighborhood of the ventricles is soft, but not fluid. The ependyma is smooth. The cerebral substance is everywhere free of blood, and rather diminished in its consistence.

After removal of the brain, the floor of the third ventricle is seen distended like a large vesicle. In looking at the base of the brain, the cerebellum, and especially the pons, seems increased in width to an extraordinary degree—the cerebellum completely covering the posterior lobes of the cerebrum. Its relief is destroyed, partly by flattening, and partly by a tumor mass, which protrudes on both sides of the medulla oblongata and the vallecula (incisura longitudinalis cerebelli). The tumor is very soft, medullary like, and excessively vascular; and grayish-red, and uniformly red spots are seen on it, so that at the first glance the differentiation of the tumor from the surrounding cerebral substance covered with highly injected pia mater, is not easy. The white pons can be distinguished from the tumor with greater ease. The glosso pharyngeal, spinal accessory, and hypoglossal nerves, as well as the facial and auditory nerves, are completely buried in the spongy mass. Not merely the tumor, but also the cerebellum, the pons, and the spinal cord are softened to such a degree that they become fluid at the least touch. It is, therefore, difficult to ascertain the precise extent of the tumor which stretches on both sides into the cerebellar hemispheres, and far into the pons. The growth makes its appearance nowhere on the surface of the cerebellum.

The lungs collapse normally. The left lung is lightly adherent at some places only, but contains air everywhere. The right lung is more closely adherent by short, tense pseudo-ligaments, and contains air in every part. The dependent parts of both lungs are full of blood and are the seat of some œdema.

In the stomach, tolerably abundant remains of food. The mucous membrane on the summits of the folds is highly injected. The transverse colon is greatly distended with gas and fæces; its mucous membrane is normal.

The liver is somewhat small; its parenchyma full of blood, brown, and in a state of commencing decomposition. The gall-bladder contains some yellowish bile.

The spleen is of normal size, soft, almost fluid.

The kidneys are very full of blood; their tissue very soft, in consequence of post-mortem hypostasis and beginning decomposition.

In the urinary bladder about three ounces of moderately concentrated urine.

—*Immermann*, Berl. klin. Wochenschrift, 1865, p. 177, et seq.

It is of course obvious that the situation of multiple neoplasms in the cavum cranii can be defined in the rarest cases only.

Diagnosis.

The diagnosis of intracranial tumors may be difficult, or, indeed, impossible; for the symptoms to which they give rise, in the incipient stage, are, for the most part, those also produced by other diseases of the central nervous system. That, however, which in the very beginning of the disease can be taken as a safe guide, is the accurately ascertained condition of the optic papilla and retina, as well as the condition of the field of vision. For the change taking place in the optic disc indicates, as a delicate manometer, the increasing pressure in the cavum cranii ; and this latter takes place, pre-eminently and at an early stage, in cases of intracranial tumors, quite independently of their position. In addition to this circumstance, the following symptoms distinguish cerebral tumors from other central diseases.

Apoplexy.	*Cerebral Tumor.*
Occurrence in advanced age, with atheroma, valvular disease of the heart, hypertrophy of the heart, contracted kidney.	Occurrence at every time of life, without any of the attendant diseases mentioned.
Without premonitory symptoms, with more or less well-marked paralysis.	After cephalalgia and vomiting; gradually approaching paralysis with simultaneous spasmodic attacks.
Muscular contractures in the advanced stages frequent.	Muscular contractures rare.

Softening of the Brain.	*Cerebral Tumor.*
Occurrence after apoplexy and other diseases of the brain in advanced age.	Occurrence with hereditary predisposition (tuberculosis, cancer) ; with tuberculosis, carcinosis, syphilis ; at all ages.
Headache not constant, and rarely severe.	Headache very common and often of insupportable severity.
Paralysis of the same side of the head and body.	More commonly, especially with basilar tumors, paralysis of the opposite side of head and body.
Prevalence of paralysis of the extremities.	Prevalence of paralysis of the face, especially with basilar tumors.

Difficulty of speech, hesitating at an early stage.	Genuine hesitating rare; when it occurs, it is most usual as a terminal symptom.
Muscular contractures frequent.	Muscular contractures rare.

ebral Abscess.	*Cerebral Tumor.*
Occurrence as the direct consequence of an injury, with otorrhœa, or with caries of the petrous portion of the temporal bone.	The result of an injury, but occurring late, often very late, in the train of symptoms.
Headache slight.	Cephalalgia severe.
Very often only insignificant symptoms, or even none.	Usually a steady increase in the various symptoms.
Violent final scene; severe meningitis.	Gradual extinguishment; intercurrent apoplexies; sometimes also meningitis.

It would of course be possible to cite here all kinds of symptoms, by means of which, as some believe, the above-mentioned diseases and others can be distinguished from cerebral tumor; we prefer, however, to incur the accusation of having been somewhat reserved in this respect, rather than the opposite. We wish to refer to two diseased conditions only, which, without doubt, might now and then be confounded with intracranial tumors, and which have occasionally been mistaken for them. Uræmia may be observed after and also along with symptoms, which render the idea of a tumor in the brain at first glance by no means absurd. Long-continued, violent headache, tumultuous vomiting, disturbance of vision, vertigo, epileptoid attacks, etc., are fully capable of favoring the deception. The true state of things, however, will become apparent as soon as the urine is tested, the heart and pulse examined, and the condition of the retina accurately investigated. Hysteria, also, gives rise to mistakes of diagnosis sometimes. An hysterical individual in particular, who has sought advice all over the world, or has occupied a bed in a hospital ward next to one in which lies a patient with cerebral tumor, may deceive even distinguished men of science.

Prognosis.

In the majority of cases the prognosis is most unfavorable. The symptoms of the disease advance steadily, and lead to death with the terminal phenomena already described. Sometimes the exitus letalis is brought about by an apoplexy, especially in cases of aneurism. Epileptoid attacks, and even simple fainting-fits, now and then have a similar result. In contrast with these cases are those in which the symptoms produced by a tumor gradually recede. Headache, vomiting, and fits cease, and even the amaurosis disappears. This may be permanent, and the patient regard himself as cured. More commonly, however, some injurious influence, *e. g.*, abusus spirituosorum, gives rise to determinations of blood to the tumor, and its increased growth. The symptoms reappear, and with greater severity, and lead to a fatal termination, either slowly, or in a few days, with violent meningitis, or without the latter.

At other times the irrepressible vomiting, the impeded swallowing, and the inflammation of the lungs, so often connected with the latter, help to hasten on the end. A similar result may be produced by bed-sores, injuries received during fits, and retention of urine and its consequences.

It is therefore useless to attempt to define the length of time over which one of these cases may extend. The commencement of the illness often dates back in the dim past, and even the most experienced pathologist will not venture to estimate the age of a tumor he may have before him. Injuries of the skull, after which the first cerebral symptoms have appeared, may afford some idea of the commencement of the disease. However, by this method it would sometimes be necessary to allot such lengthened periods—so many years—for the existence of a tumor, that one might begin to doubt the accuracy of one's observation, were it not that Lebert has reckoned the average length of growth of a cerebral carcinoma to be one and a half years, the longest five years, and the shortest three months.

Treatment.

Even in this so hopeless territory the treatment should not be regarded from a nihilistic standpoint; and we would not recommend, in a general way, that all curative attempts should be abstained from in these cases, as has been done in some quarters. We would remind the reader that injuries to the cranium very frequently give rise to intracranial tumors. Now if these injuries, as a rule, run so much worse a course, the more other determinations of blood to the skull take place, it may certainly be accepted that a new-growth will follow upon such a trauma all the surer the more irritation the brain is subsequently exposed to. Hence individuals who have suffered from a head injury, with lesion of the bone, or with cerebral symptoms, must be kept under the strictest control for a length of time afterwards. Everything should be avoided which would excite an increased flow of blood towards the head, and the occupation and mode of life should be regulated accordingly. Above all things, alcoholic drinks and other irritants of the brain must be strictly abstained from, and sleep, which reduces the quantity of blood in the brain, must be provided for in sufficiency. The other functions of the body should be carefully regulated; irregular action of the heart, disordered digestion, etc., should be corrected. If, notwithstanding all this, incipient symptoms of cerebral tumor appear, it will be necessary to interfere according to rational indications.

For almost all cerebral tumors the presumption is true, that local hyperæmiæ play a part in their production. It is therefore perfectly right that we should treat these incipient symptoms as local hyperæmiæ, especially when a cranial cicatrix makes the situation of the latter probable. If the affected individual be full-blooded, and the symptoms violent, the application of leeches to the nasal septum and behind the ears may be recommended. In any case, the skull, or the injured region of it, should be covered with iced compresses, and the most careful regulation of the diet and purgation of the bowels be attended to.

Should the symptoms affect an individual in whom a hereditary predisposition to tuberculosis exists, or perhaps tuberculosis be already present, it will be necessary to keep such a one, who is generally doubly excitable, removed from all excitement; and, of course, all debilitating methods of treatment should be avoided. The same rules hold good where there is a suspicion of carcinosis. With persons who have suffered from syphilis and who still manifest symptoms of this disease, an antisyphilitic treatment should not be neglected. Hesitation in this matter may entail serious results; for what cerebral syphilis has once destroyed, can never be repaired. An active course of inunction, of iodide of mercury, or iodide of potassium, cannot, therefore, be spared in these cases.

Of the more active derivatives in common use, we prefer the seton to the application or inunction of the ointment of tartarized antimony. There can be no doubt but that resorbable deposits are excited to a more rapid resorption by the exfoliation of a large piece of integument and of bone of the skull, which one often sees after a thorough rubbing in of the above ointment; and all the more so if the process be attended with erysipelas and fever. The method, however, is most annoying to the patient, and its effect very difficult to foresee, so that we adopt it in only a few special cases.

It is much easier to apply the seton and to limit its effects, and consequently it is better to make use of it in persons who are not too irritable or reduced in general health. One now and then may observe an undoubted improvement after it.

We have little to say concerning other means for directly influencing the growth of a tumor. Iodide of potassium—which has the frequent complication with syphilis, no doubt, to thank for its reputation—is often tried. A trial of it should not be neglected. Wunderlich thought that the long-continued use of small doses of arsenic sometimes had a beneficial effect. In some cases in which we tried it we did not find it of much use; but the method requires to be further tested.

With regard to the symptomatic treatment, the cephalalgia deserves our first attention. Narcotics cannot be dispensed with here, and subcutaneous injections of morphine will be found of most service.

Bromide of potassium sometimes performs good service where there are frequent convulsive or epileptic attacks. Paralytic conditions, where they are slowly progressive and accompanied with few symptoms of irritation, may be treated with electricity. For reasons which cannot be explained here, the constant current is the most suitable for use; but it should not be applied for too long a time, or in too great strength.

Constipation should be treated with bitter mineral-waters and other means, while the condition of the stomach and general nutrition is attended to. For the attendant amblyopia, amaurosis, and hemiopia, even the oculist can do nothing; still we should not neglect to have him examine the condition of the fundus oculi. A fungus duræ matris, which has grown through the skull, is simply to be kept clean and protected from injury and from surgical interference.

SYPHILIS OF THE BRAIN

AND

NERVOUS SYSTEM.

HEUBNER.

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History.

The exacter knowledge of syphilis of the nervous system dates only from the last few decades. The attention of the earliest observers, in the first century after the appearance of syphilis, was so strongly directed to the external symptoms of the disease, which broke out in frightful intensity, that the idea of analogous effects in the internal organs appeared only in occasional hints.

Although Fracastor described so graphically the ravages of syphilis in the face, etc., he knew nothing of the internal diseases of the same patients. And although it was known to Ulrich von Hutten that syphilitic patients occasionally became paralyzed, he referred the trouble not to the disease, but to its treatment. Here and there—for example, in Nicolaus Leonicenus—mention is made of internal pustules or the rising of syphilitic vapors to the brain, but no accurate data are given as to internal syphilis, or syphilis of the nervous system in particular. Attention was first called to the injuries which the brain received externally from caries of the bones; Botalli and others were aware of such cases.

The first description of a syphilitic gumma of the brain comes, so far as I can discover, from Ballonius, who announces, as a curious and rare case, the discovery in the corpora mammillaria of "abscessus, quos melicerides vocare possis, quamquam ad scirrhum proxime accedant," and refers them to concealed syphilitic poison.

This observation dates from a period previous to 1616, in which year Baillou died.

During the remainder of the seventeenth century we meet with very scanty anatomical data—as, for instance, in Guarinoni (also mentioned in Bonet's Sepulcretum), who uses the name gummata, and also in Morgagni.

Certain nervous diseases, as epilepsy and neuralgia, are somewhat more frequently referred to syphilis and its latent poison as causes, although these views are not anatomically sustained.

This poverty of data, in comparison with the great number of writers who busied themselves for so long a time with syphilis, is not to be wondered at, if we reflect how few physicians practised post-mortem examinations, and that the great majority concerned themselves only with symptomatology, and especially with discussions on therapeutics.

But even when symptoms alone were considered, the remark could not fail to be made that nervous troubles (as well as others) of a peculiar character were extremely frequent in syphilitic persons; and thus the idea of a latent or transformed syphilitic poison, which expended its force upon the internal organs, constantly gained ground. In Astruc's great work we find whole chapters devoted to a great number of syphilitic nervous diseases; but although this physician endeavored, at least in part, to find an anatomical basis for the symptoms, later writers—for instance, Carrère—took no further trouble about it, but simply stated that any disease, and of course any nervous disease, if it lasted long enough, might be considered the effect of a disguised or masked syphilis, and be treated accordingly.

Against this view the weighty voice of Hunter[1] was justly raised—who indeed, since he ascribed susceptibility to disease only to certain tissues, doubted whether the internal organs, and expressly the brain, could be affected by syphilis. In general, at the beginning of this century, similar views were held by the majority of physicians. As so frequently happens, a fact, correct in itself, was generalized upon in so unscientific a way, that it was entirely rejected by sober heads, and then gradually, freed from exaggeration, again resumed its true importance. Especially in France, in the early decades of the present century, scattered cases of syphilis of the brain and nerves were frequently reported, supported with increasing frequency upon trustworthy autopsies.

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[1] Treatise on the Venereal Disease. 1787. Part 6. Section 7.

Lallemand, Rayer, and others were active in this direction; and since the doctrine of syphilis in general had been made clear by Ricord, these reports of internal syphilis gained in credibility. After Schützemberger, in Strassburg, and his school had published a series of studies on cerebral syphilis, attention was again directed to this point in Germany and England. Dittrich's description of syphilis of the liver, and especially Virchow's anatomical investigations (published in 1858), contributed not a little to open the eyes of skeptics to the existence of internal syphilis. From all sides came observations upon the syphilitic new-formations in the skull, in the dura and pia mater, in the brain, and upon simple inflammations and softening of the brain in syphilitic patients, and finally, since 1854, but especially within the last ten years, upon syphilitic disease of the arteries of the brain. Reports of anatomical changes in the spinal cord and the peripheral nerves were more rare, but not altogether wanting.

Besides the anatomical investigations, upon which indeed everything depended, more careful study was now directed to the phenomena of nervous diseases in syphilitic patients. And the fact, mentioned by the older writers, but since almost forgotten, was again noticed, viz., that syphilis could give rise to mental disturbance, epilepsy, neuralgias, and paralysis. The mere fact, however, was no longer sufficient, but anatomical reasons therefor were sought.

This path was marked out by the monographs of Leon Gros and Lancereaux, and of Zambaco, which appeared in 1861 and

1862, and soon after pursued in Germany by Jaksch, who found numerous followers during the last decade in many places, especially in England. At present it seems that syphilis of the nervous system, as being comparatively the darkest domain of internal syphilis, is precisely the point which, within the last few years, has most attracted the attention of physicians.

Etiology.

It is chiefly from the etiological point of view that it is both correct and easy to speak collectively of such various, and, apparently, dissimilar diseases as the nervous system presents when affected by syphilis. Anatomically, also, the identity of the disease in individual cases, however different they may at the first glance appear, is becoming more probable, but cannot as yet be expressed in general terms ; while, as regards the symptoms, certain characteristic marks of syphilitic nervous affections may be stated, though all the cases will not correspond to the description. Etiologically, however, all diseases of this kind meet at one point: constitutional syphilis must be present in the body, the nervous system of which is specifically diseased.

This self-evident proposition relieves us of the trouble of inquiring into the essential cause of these affections; we have always to do with a secondary disease, which begins in consequence of a constitutional affection. As to the more intimate connection between the latter and the outbreak of a syphilitic nervous disease, we know scarcely more than did the older writers. Like them, we must assume a poison which is seated in some part of the body, from time to time gets into the circulation, and acts as an irritant, now here and now there—in our case specially on the nervous system.

Some of the more precise conditions under which the nervous system is particularly affected may be here mentioned:

1. The position of nerve syphilis in the progressive series of syphilitic diseases. It belongs, like the corresponding affections of the liver, the heart, etc., to internal syphilis, and as such, in general, to the later secondary, or, if one prefers, tertiary forms.

The distinction between secondary and tertiary, so long as the inoculability of the one, and the non-inoculability of the other are not finally settled, is of little consequence; for, anatomically, the majority of brain and nervous affections are just as much equivalent to the indurated chancre as are the gummata of the skin and bones. It remains, therefore, in order to get an idea of the position of nerve syphilis, to state the length of time which has elapsed, in as many as possible of the cases heretofore described, between the first infection and the appearance of the nervous disease, and to notice the intervening specific symptoms.

The latter is specially important, since the rapidity with which syphilis spreads, so to speak, over the whole body is exceedingly various in different individuals and under different methods of treatment. It must also be considered that the nervous system may become diseased in very different ways under the influence of syphilis, and that the various forms may possibly in point of time occupy different places in the course of syphilis. I have made a comparison, from these points of view, of the most trustworthy cases accessible to me.

By far the greatest number of cases refer to diseases depending on syphilitic new-growths. I include here, for reasons which will be elsewhere given, the cases with disease of the arterial system of the brain.

Among forty-five cases of so-called syphilitic tumors in the membranes of the brain or spinal cord, the nerves, etc., the time since the first infection is stated fifteen times. It is almost always over one year, in the majority of cases over three, in two cases thirty and over thirty, and these are just the cases in which the other symptoms of syphilis are not mentioned.

Only twice in the fifteen cases did the affection begin in the first year, and as in these nothing is said in the report of the autopsy, as to other evidences of internal syphilis, they may be looked upon as exceptional (one case of Faurès in Lancereaux and Leon Gros, l. c. Obs. 137; one case of Engelstedt, l. c. p. 144).

In nine of the forty-five cases a period of several or many years is spoken of, and in ten other cases the traces of old syphilis were found post-mortem in other places, viz., in the liver, bones, and skin (gummata).

Among twenty-four cases of arterial syphilis, partly with and partly without other new-formations in the interior of the skull, where data referring to this point are given, the disease began once nine months after infection; once two years; fifteen times three or more (up to twenty) years intervened between infection and the beginning of the brain trouble; six times long-continued syphilis is spoken of; and once, where the time is not stated, old cicatrices were found in the liver.

Among twenty cases of simple inflammation of single portions of the brain (among which, perhaps, are many cases of cerebral softening from arterial disease), the duration of the constitutional affection, up to the outbreak of the disease, is mentioned sixteen times. In five cases it was less than a year; in three less than one and a half years; in eight cases several years.

From these figures it appears that when a syphilitic growth is developed in the nervous system, the disease almost invariably appears at a late period—in fact, when the syphilis has lasted for several or often many years.

This rule, of course, as some of the above cases show, is not without exceptions. Development of a gumma in the brain may take place even at the period of the early secondary symptoms. In these cases, however, considering the great rarity of the occurrence, the co-operation of certain accessory causes may be assumed.

Simple inflammation (or softening) seems to occur more frequently than cerebral new-formations in the early stages of syphilis; at least, we find among our cases a somewhat larger percentage of early inflammation of the brain, a full half happening within two years; but since, on the other hand, we meet here with cases which appear for the first time long (three times twenty years, once seventeen years) after infection, it may be asked whether, among the different forms of simple syphilitic softening of the brain, we are not in reality dealing with several varieties which ought to be further distinguished. I may at least call attention to the question in reference to this little studied syphilitic disease.

In general, however, syphilis of the nervous system, like that of the bones and liver, may be reckoned among the later secondary or tertiary affections. This is not to say, however, that every patient suffering from syphilis of the nervous system has previously undergone a great number of the earlier affections. In many reports of autopsies, indeed, we find mention of cicatrices in the skin, bones, liver, etc.; but in many others, to all appearance careful ones, these data are wanting. In twenty cases out of forty-one, Engelstedt found the syphilitic nervous disease the only symptom; but in the other half syphilitic eruptions or their remains were to be seen. The same author reviewed his cases, to determine whether repeated relapses specially favored the outbreak of nervous syphilis, and found that, among thirty-five patients with syphilitic nervous diseases, only eleven had had recurrences of constitutional syphilis.

On the other hand, an incomplete, hesitating treatment of external syphilis specially predisposes to affections of the nervous system, as well as to other internal troubles.

2. *Predisposing causes.*—Paracelsus has already stated that syphilis takes in every man the character of that disease to which he is inclined by hereditary or other predisposition. Virchow has correctly called attention to the fact that the localization of syphilis is determined by external injuries and accidents. If this is true of the skin, bones, etc., it may be assumed for the nervous system.

Attention seems not to have been particularly called to this point, most authors having been contented with recognizing the most essential cause of the nervous disease. The knowledge of accessory factors, however, is not only of general interest, but also of practical importance in the particular case. Lagneau, Lancereaux, and Engelstedt mention some of these accessory causes.

Hereditary predisposition to nervous diseases, to the importance of which in reference to all diseases of this class Griesinger has called attention, undoubtedly plays an important part. It is highly probable that an organism, whose nervous system is already by inheritance in a condition of unstable equilibrium, may be affected by nerve syphilis both easily and at, perhaps, an early period, when subjected to infection. Some cases of this kind, though rare, may be cited.

Lallemand (l. c.) speaks of a patient who was subject to migraine from youth, and died of cerebral syphilis eight years after infection; Zambaco (l. c., Obs. 55), a syphilitic softening of the brain in a patient whose father died of apoplexy, and who had himself suffered for years with congestion of the head. In one of my cases a patient, who afterwards died of cerebral syphilis, had had, as a child, essential paralysis. In a second, of similar character, the father had been subject to congestion of the head, with eccentric behavior, a brother was mentally feeble, and he himself had suffered with headaches from his youth. A case of recovery which came to my knowledge was that of a patient whose father was apoplectic and brother epileptic.

Traumatic influences may favor the outbreak of nervous disorder in syphilis, not only by exciting disease of the bone, with caries and its consequences, but also, by direct irritation, leading to the formation of gummata or inflammation on or in the nerve substance.

In Lallemand's case, already mentioned, the nervous symptoms appeared soon after a blow on the head. Rul Ogez[1] saw severe cerebral disease follow a blow upon the head with a stick, in a person formerly syphilitic, and cured it with iodide of potassium. In one of my cases (l. c., Obs. 47), a severe wound had preceded the symptoms. Wilks and Wagner report similar cases, in which, however, the wound had long preceded the syphilitic infection.

Psychical influences are just as important: severe mental labor, fright, excessive bodily exertion, great excesses, with their consequences.

It may easily be imagined that by causes of this kind disturbances of the circulation, congestions, paralyses of the blood-vessels, or stoppages of the blood may be produced in the brain, the meninges, or the cranial periosteum, and that the syphilitic virus already present may influence such places with special ease. Engelstedt cites a number of cases where excessive study, excessive venereal indulgence, etc., gave rise to the occurrence of cerebral syphilis.

Joseph Frank[2] treated a woman who had become epileptic after a fall into the water. He found on the skull a syphilitic destruction of the bone, and cured the patient by an anti-syphilitic treatment.

Mildner (l. c.) saw in one case (Obs. I.) a syphilitic psychosis arise in consequence of excessive mental labor.

The fact stated by Lancereaux, that more cases of cerebral syphilis occur among the learned professions than in the lower classes, may very possibly be connected with this condition—that is, the demand for great exertion of the brain.

An earlier view, embraced by Ulrich von Hutten, and not entirely abandoned by Lancereaux and Leon Gros, that mercury, used as a cure for syphilis, could produce softening or inflammation of the brain, must be given up, since the symptoms and anatomical changes of mercurialism have been determined by Kussmaul's admirable investigations, and found to be of an entirely different character.

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[1] Gazette Médicale de Paris. Juillet, 1843.

[2] Praxeos medicæ universæ præcepta.

3. Individual conditions have no weight in the etiology of this affection. Nerve syphilis occurs at every age at which the constitutional affection can be present. In about one hundred cases, accessible to me, the number of years reaches up to sixty-eight and down to twenty-two. But the first year of life is not spared if a child suffers from hereditary syphilis. Graefe[1] describes the case of a child less than two years old, in which a syphilitic new-formation was found in two cerebral nerves, besides a softening in the brain.

The sex is of just as little importance. The fact that fewer women are thus affected (in my cases, for instance, sixty-four men, twenty-eight women) depends upon the relative infrequency of venereal diseases in general in the female sex.

Constitution, nourishment, and other external conditions, may have some influence upon the progress of the disease, but not upon the disposition to its formation.

Pathology.

In describing the morbid appearances and symptoms of the disease with which we are concerned, it is no longer possible to treat the whole subject at the same time. Considering the great variety of the seat, the distribution and the character of the abnormal processes, it would only lead to confusion if a general description of the disease were made up from the history of the cases reported, as has generally been done in the current abstract representation, even in the works of Lancereaux and Lagneau, for example, where the symptoms are stated chiefly according to the frequency of their occurrence in one or another form of disease, but are not thoroughly investigated in their anatomical and physiological relationships. An attempt to supply this defect, even if incomplete, must be made here, if our insight into the nature of these diseases is to be increased by further observations.

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[1] Arch. für Ophthal. I. Bd. 1 Abth.

We will consider, separately, syphilis of the brain, the spinal cord, and the peripheral nerves; and here, again, shall have to distinguish, anatomically and clinically, several individual forms and localizations of the disease.

Syphilis of the Brain and its Membranes.

Pathological Anatomy.

a. *Syphilitic new-growth.*

The most frequent change met with in the cranium of patients, who develop cerebral disease under the influence of syphilitic cachexia, is the formation of a heteroplastic tissue, the syphiloma or gummous tumor. We shall therefore describe it first. The new-growth appears in two very different forms, which, however, are often united in the same body, either (first) as a whitish or grayish-red or moist gray mass, of the consistency of a firm jelly, in thinner places half translucent; of irregular form, generally determined by the place where it is seated; discharging upon its cut surface a scanty whitish-red juice, and gradually blending with the surrounding tissues; or (second) as a yellow, firmer—often as firm as cartilage—dry, friable substance, upon section homogeneous and cheesy, which is found either in the form of somewhat sharply circumscribed larger or smaller tumors, or invading the grayish-red substance just described, sometimes as larger nodules and streaks, and sometimes as a more minute marbling.

The grayish-red mass consists chiefly of round cells, with a small proportion of nuclei, and, among them, spindle and stellate cells, arranged sometimes apparently without order, and sometimes forming groups together with an alveolar framework. These cell masses lie, as far as possible, in the interstices of the original tissue, the constituents of which, altered or not, form the chief part of the intercellular substance of the new-growth, which is permeated by scanty capillaries, often of large calibre. Hence in places where the original tissue was delicate and contained much water, the growth is softer (subarachnoid space); where it was more dense and fibrous, firmer (dura mater). Where the interstices of the tissue are large, there numerous cells lie together in large alveolar spaces, which may be again divided into secondary spaces by finer fibres (Wagner); where the meshes are small, only a few cells lie in narrow fissures. In the first case we have to do with a growth rich in cells; in the latter, with one more fibrous and brawny. The source of the chief mass of the cells is probably to be found in the blood; the granulation cells have mostly emigrated; the spindle cells, which in many places are arranged in a more organized structure, as young connective tissue, in the meshes of which round cells lie, probably arise from the connective-tissue strata of the place at which the growth originated. One can often observe how the same growth, which in one place has the character of indifferent granulation tissue (for instance, in the subarachnoid space), upon reaching another tissue (for instance, brain substance), immediately assumes an exquisite alveolar structure. In tumors of the subarachnoid space the mass of cells lies imbedded in a gelatinous intercellular substance within the meshes of connective tissue.

The whole new-formed tissue is permeated by blood-vessels, chiefly of a capillary character, which are more or less abundant according to their abundance in the original structure; in some places little extravasations may be found, which mark the cut surface of the new-growth with reddish points. Many of these blood-vessels, especially the smallest, possess a much-thickened wall (perithelium).

This form of tumor is never sharply defined; on examining the edges by the microscope, one may see at points apparently normal a strongly marked cellular infiltration, which gradually passes into the healthy tissue.

The yellow mass is found first in the form of veins, streaks, or so that one or several yellow dry nodules surrounded by the grayish-red substance, project into the structure just described. This is particularly frequent where the new-growth passes into nerve tissue, or is chiefly situated therein. It then consists either (1) of a dry, somewhat shining fibrous substance, appearing in thick sections, under the microscope, gray or grayish-yellow, the origin of which cannot with certainty be determined, but which may be in some cases the remains of the original tissue compressed by the new-growth (atrophic neuroglia), and, in others, masses of atrophic cells of the neoplasm itself; or (2) (especially in nerve sheaths, in the tissue of the dura mater, in the larger dry masses) of cells, round or spindle-shaped in a state of fatty degeneration—that is, masses of granule cells, which are situated in large agglomerations within the meshes of the original tissue.

Secondly, we find them in the form of completely circumscribed, often almost encapsuled tumors (like cerebral tubercles), around which remains of chronic inflammation, or the gray-red new-formation may be found. They are as large as an almond or dove's egg, and in the first case several often lie together. Their form is not unfrequently determined by the space in which they are seated (fissures between cerebral convolutions), behaving thus simply like a caseous inflammatory exudation. Upon microscopic examination it seems that this homogeneous mass consists of a granular substance, entirely uniform upon thin sections, apparently without further structure, and completely devoid of vessels. When broken up or torn apart, however, it sets free for the first time a multitude of angular or roundish granular elements, which resemble shrivelled or broken cells of roundish shape, and may correspond very well to the remains of the round cells which form the grayish-red syphiloma. Of the other elements, connective-tissue fibres, spindle cells, etc., nothing more is to be seen. Here and there lie groups of needle-shaped crystals of fat, with granules and crystals of blood pigment intermixed, while nearer the edges of the yellow growth may be found upon section masses of fatty granulation cells. The chief part of the process, however, here, does not consist in fatty degeneration, but in genuine cellular atrophy (caseification, tubercularization, in the French sense). In the interior such tumors are perfectly dry, or some small spots of softening may be found. The surrounding tissue always shows a recent, tolerably vascular, proliferating cell-mass, like that which we find in general in the grayish-red growth; and even where such a mass is encapsuled (which is always the case with the tumors situated in the dura mater), the meshes of the connective tissue forming the capsule are infiltrated with young cells.

It is probable, if not yet strictly proved, that these yellow tumors originate in the grayish-red, and represent the dead and atrophied remains of the younger soft tissue.

This neoplasm in its various forms has an entirely different signification, and even certain anatomical peculiarities, according to the locality within the cranial cavity in which it is developed.

It has two favorite seats: the dura mater and the subarachnoid space.

The growth very frequently affects the dura mater alone, without the participation of any other tissue in the cranial cavity. It then develops between the two (greatly thickened) layers of the dura mater, and is beautifully encapsuled thereby.

The soft grayish-red growth is almost never met with in this place, but either the fibrous modification of the cellular neoplasm, or, most frequently, the dry, yellow tumor, which may attain the size of a dove's or even a hen's egg. It may be developed in any region of the dura mater, but grows particularly large in the duplicatures, such as the falx, etc.

One frequently meets with smaller, recent, but yet firm tumors in the dura mater overlying the sphenoid bone. In such cases the membrane is thickened around the tumor and for some distance in the neighborhood, and the interstices of the connective tissue infiltrated with rows of round cells; the part of the cranial bones corresponding to the tumor is subject to the so-called caries sicca, is rough and eroded; the rest of the cerebral contents are normal. The effect of all these tumors is to diminish the capacity of the cranial cavity, and to produce tension of the dura mater.

The consequences for the whole neighborhood are much more important when the neoplasm is developed in the subarachnoid space. Under these circumstances all the surrounding tissues are affected—all the membranes, all the organs (vessels and nerves), which traverse the space, and the brain itself. The majority of all cerebral syphilomas originate on this surface, and grow from the subarachnoid space and the pia mater towards the brain substance. This fact, already stated by Wilks, Jaksch, and others, is confirmed by a comparison of the quite numerous cases collected by me. Any portion of this space may serve as the starting-point. It is only occasionally determined by a lesion of the bone, for instance, while usually it cannot be ascertained why any particular spot undergoes syphilitic degeneration.

If the disease is seated in the subarachnoid tissue which surrounds the cortical substance, where the dura mater lies closer to the brain—that is, on the convexity and the lateral surface of the brain—we usually find the following arrangement: The dura mater is closely united to the surface of the brain to an extent of from one to perhaps two inches square, very much thickened at this point, often with a yellow translucency. On removing the dura mater, this portion must be left adherent to the brain, or the place will be lacerated. If a section is made perpendicularly into the brain through the dura mater, it is seen that the latter, the arachnoid, the subarachnoid space, pia mater, and surface of the brain, are blended into a single mass, of which the different constituents can no longer be distinguished. A moist reddish layer appears between the outer surface of the dura mater and the brain, within which, corresponding chiefly to the depressions formed by the beginnings of the sulci, lie one or several dry yellow growths as large as beans. These tumors generally run into the surface of the brain, and no sharp boundary can be drawn, as a more or less extensive portion of the cerebral cortex and even of the medullary substance is changed into a dry, yellow mass. The brain substance surrounding the yellow mass shows a white or red softening, which is often extensive, and may spread over half a lobe or more.

If the affection is seated at the base, the dura mater participates less frequently, and the neoplasm is then prone to fill the spaces around the chiasma, the infundibulum, between the crura, at the anterior or posterior border of the pons, which are bridged over by the arachnoid.

In this region the grayish-red growth is usually met with, which also spreads into the nerve substance to a varying depth, —often completely grows through the chiasma, includes the hypophysis, grows into the cavernous sinuses, and then again leads to circumscribed softenings in the nerve substance.

In both regions, however, the new-growth may completely lose the character of a circumscribed tumor, and take that of a diffused infiltration. In such cases we may find, at the base of the brain, a gelatinous gray exudation, stretching from the roots of the olfactory nerves to the cerebellum, and resembling many forms of tubercular meningitis, except that the exudation here has the structure of a syphiloma. In fact, the term gummous meningitis is more appropriate than syphiloma of the brain. The same thing sometimes happens on the convexity. All the membranes over one or both hemispheres are grown together into a more or less thick brawny mass, which, upon more careful examination, resembles throughout the structure of a syphiloma. The surface of the brain is then extensively softened. Often, under some methods of treatment, the new-growth gets well and a cicatricial thickening remains over the hemispheres, which in the dead body may be erroneously spoken of as the remains of a simple pachymeningitis.

Only in rare cases the syphilitic growth seems to make its appearance, in the form of numerous miliary nodules, upon the dura mater, or in the soft membranes. Engelstedt[1] describes one such case, as well as Leon Gros and Lancereaux.[2]

Tumors in the interior of the brain, without connection with the membranes, belong among the greatest rarities. Since the question of the origin of syphilitic tumors from the brain substance is of general pathological interest, I will here adduce the accessible cases.

Among forty-five cases of syphilitic new-formations in the cranial cavity, with accurate statements as to their seat, I find only three of tumors of the brain substance, which neither lay upon the surface of the brain nor were connected with it. They are the following:

1. One case of Lallemand,[3] where a tumor, as large as a small nut, was situated in the centrum ovale of Vieussens, surrounded by softening. To say nothing of the case being somewhat doubtful (the symptoms were very peculiar), it is not difficult to suppose an unperceived connection with a cerebral fissure. Tüngel[4] describes a similar case of tumor in the centrum ovale, which, however, was connected with peripheral tumors.

2. A case of Zambaco,[5] where, besides a gumma, as large as a dove's egg, on

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[1] l. c., p. 146.

[2] l. c., Obs. 124.

[3] Union médicale. 1853, p. 441, in *Leon Gros* and *Lancereaux*, l. c., p. 254.

[4] *Lancereaux*, Traité de la Syphilis, p. 474.

[5] l. c., Obs. 84.

the surface of the pons, a smaller tumor, surrounded on all sides by brain substance, was found in the left optic thalamus.

3. A case of Lancereaux,[1] where tumors, as large as peas, consisting of fat granules, and surrounded by a connective-tissue membrane, were found in the corpus striatum.

Of these last two cases, Lancereaux's is not quite conclusive, since it may well be doubted whether this mass of fat granules actually originated in a syphilitic growth; and in Zambaco's case no histological examination is reported. In the rest of the cases, mentioned above, the lesion was seated at the periphery, so that, among all the carefully described cases which were available for analysis, there was not a single one in which the seat of the tumor was undoubtedly in the interior of the nervous substance.

In this place should be mentioned a case, apparently unique, described by Faurès,[2] where a girl died somewhat suddenly during the existence of recent syphilis, and a raspberry-like growth, resembling the erectile tissue in structure, was found in the fourth ventricle. Here, of course, the growth originated in the reflection of the pia mater.

Besides the places already mentioned, the syphilitic new-formation establishes itself at one other, which is of the greatest importance for the vitality of the brain—that is, in the interior of the great arteries at the base. I have described this peculiar occurrence with considerable elaboration in the work cited above, and must refer to it for the details. Fifty cases of this form of disease (of which some indeed are doubtful) are there collected. In addition to the atheromatous process, which sometimes affects the vessels of syphilitic patients, the arteries at the base of the brain are subject to a peculiar form of disease, sometimes when a syphilitic growth is developed around them, but sometimes entirely independent thereof.

As the disease begins, the blood-vessels become less transparent, their reddish color gets a whitish glimmer, and finally becomes completely grayish-white; they lose their usual shape of a flattened cylinder, and become completely round; they are firmer to the touch, and at last perfectly stiff and hard as cartilage. Upon section, we may remark, even with the naked eye, that the lumen of these vessels has been more or less encroached

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[1] Traité de la Syphilis, p. 459.

[2] Comptes rendus de la Sociéte de Toulouse. 1853. *Leon Gros* and *Lancereaux*, l. c., p. 256.

upon—at first by semi-lunar segments, and later by zones of new-formed substance lying around the periphery. This substance is of a white or gray color—at first moist, afterwards more dry, of a tough, in the later stages hard and cartilaginous consistency. The calibre of the vessel is thus reduced to the third, fourth, or fifth of its original dimensions, and, at last, places may be found where the remaining passage is stopped by an adherent thrombus, or where there is no passage at all, but the whole artery is changed into a solid cylinder. In such cases the whole arterial system at the base of the brain exhibits a general narrowing and shortening.

This new-growth is developed between the elastic lamina of the intima (membrana fenestrata) and the endothelium, and consists at first of endothelial cells, which constantly increase and change into a firm, felted connective tissue, composed of spindle and stellate cells, into which the emigration of round cells from the vasa nutritia of the vessels takes place, so that a granulation tissue is formed like that of syphiloma in other places. The new tissue grows often in distinct periods, in two directions, towards the interior of the artery, and in the direction of its length; and thus, on the one hand, gradually narrows the starting-point, and, on the other, involves in the degeneration more and more of the main artery, with the communicating vessels and branches, even to those of small dimensions.

In its further course the new-growth either becomes organized—taking a similar structure to that of the original wall of the vessel, and the process comes to a standstill after great diminution of the vascular calibre—or cicatrization occurs after complete obliteration with transformation into fibrous connective tissue, and the whole portion of artery affected becomes useless.

By this process a more or less extensive portion of the channels by which blood is carried to the brain is rendered materially less capable of its normal action, and functional disturbances of the brain substance arise; or when, either by the new-growth itself or by an added thrombus, such portions of the vessels as give rise to terminal arteries are obliterated, softening of the brain takes place. This degeneration most frequently affects the carotids and their branches, the arteries of the fossa Sylvii, and of the corpus callosum, near their origins. But it is exactly these portions (see "Die luetische Erkrankung der Hirnarterien," 4. Capitel), from which the terminal arteries supplying the nucleus lenticularis and nucleus caudatus[1] arise; and hence the frequent softening in these regions. In general a large, perhaps the largest, part of the so-called syphilitic inflammations or softenings of the brain are to be referred to this degeneration of the large arteries.

The softenings in the neighborhood of syphilomata and in gummous meningitis are chiefly consequences of the degeneration of the small arteries situated in the region of the syphilitic neoplasm.

Hence, when syphilitic inflammations are developed in the brain, it is necessary, even if the large arteries at the base are apparently sound, to examine the condition of the small vessels penetrating from the surface, since the degeneration may possibly be developed in them alone.

b. *Syphilitic inflammation.*

Besides the cases of undoubted new-formations in the interior of the cranial cavity, we meet with many recorded observations relating to the presence of non-specific inflammation or softening, partly of the membranes, partly of the brain substance, which are, however, referred to a syphilitic cause, because they made their appearance during the existence of the constitutional disease, or even simultaneously with the recent external eruptions.

Those cases which depend upon the extension of a suppuration of bone to the dura mater and brain, must be at once excluded, since they belong here only remotely, and fall under the category of cerebral abscesses of external origin, with the difference that here the septic inflammatory process arises from a bone which became carious from syphilis, instead of in connection with tuberculosis, etc., as in other cases. On the other hand, the cases of primary inflammations arising within the cranial cavity are of special interest; for if the syphilitic poison can give rise to ordinary irritative processes as well as to a peculiar new-formation, any anatomical characterization of its action becomes very uncertain, and a simple conception of the disease we are considering—which has just received so powerful a support from the proof of the morphological identity of the hard chancre with all other gummata—is rendered almost impossible. Hence an analysis of the cases bearing on this point becomes necessary. In all they are much fewer than those of gummous new-formation; among some one hundred and fifty cases, we find descriptions of simple inflammations about thirty-six times—that is, in from one-fourth to one-fifth of the observations. I have assembled twenty-two of the most trustworthy cases.

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[1] Forming together the corpora striata.

Here are to be found, first, cases of simple meningitis. Mildner[1] mentions a case where a patient with a recent syphilitic exanthema, suffered from mental disturbance, progressive dementia, and an apoplectic attack, and the autopsy disclosed a pachymeningitis on the left side with atrophy of the brain.

Ljunggren[2] another, where epilepsy and dementia began twelve years after infection, and were found to depend upon simple meningitis. Griesinger describes the course of an acute psychosis of peculiar character in a patient infected not long before, where the autopsy showed a great (one-half line) thickening and tendinous toughening of the pia mater with alteration of the vessels at the base, the brain substance itself being without marked abnormality.

These cases of apparently simple meningitis cannot be looked upon as conclusive for the occurrence of non-specific inflammation, because in all three the histological examination is wanting.

I have stated above that the remains of gummous meningitis may give to the naked eye exactly the impression of connective-tissue indurations, especially as the microscopic characters of the two are the same. The formation of the thick and toughened patches, which Griesinger describes, is not common in ordinary inflammation of the soft membranes, and probability in this case rather inclines to the side of actual gummous inflammation.

More numerous cases are reported of softening and inflammation of the brain. We find mention made of foci, sometimes yellow, sometimes of a red, dregs of wine color, or having small apoplexies scattered through them, or of single parts of the brain of a whitish, mushy character. The consistency is spoken of as sometimes semi-fluid, sometimes merely soft, sometimes like bacon. Separate harder portions among the soft are often mentioned, as well as the formation of cavities in the soft parts. Their seat, when in the substance of the hemispheres, was upon the surface (in four cases out of nineteen; Gubian in Leon Gros and Lancereaux, Obs. 115; Engelstedt, l. c., page 136; Lallemand, Leon Gros, and Lancereaux, Obs. 116; Steenberg, l. c., Obs. 27), in the ganglia alone four times; in the corpus striatum (Ljunggren, l. c., Obs. 6 [cyst], and Obs. 3 [apoplexy]; Steenberg, l. c., Obs. 3 and 4); once in the optic thalamus (Faurès, Leon Gros, and Lancereaux, Obs. 105); three times in both ganglia and hemispheres (Leon Gros and Lancereaux, Obs. 111 and 112, Oedmanson); once laterally from the lateral ventricle (Flemming in Leon Gros and Lancereaux, Obs. 126); once in the cerebellum (Gjör, ibid., Obs. 88).

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[1] l. c., Obs. 5.

[2] l. c., Obs. 18.

Three times softening is described upon the surface of the brain, with adhesive meningitis (Steenberg, l. c., Obs. 16, Obs. 35; Zambaco, l. c., Obs. 52); and, finally, a general softness of the brain substance is mentioned (Leon Gros and Lancereaux, Obs. 114; Steenberg, Obs. 29; Mildner, Obs. 4).

The objection may be made to these cases that in none of them was the character of the inflammatory process established by careful histological examination. This is the more imperatively demanded, since in several cases the macroscopic description does not agree with the ordinary appearances of so-called brain softening. Thus, Lancereaux speaks in his 112th Observation of a yellow apoplectic focus in the corpus striatum. Zambaco, describing a similar case, says that a grayish nucleus was situated in the softened mass. In Lallemand's case a purulent focus is spoken of, having tubercles as large as grains of barley situated around it. Fleming describes the medullary substance as lardaceous. In Steenberg's fourth case the softened spot in the corpus striatum is spoken of as grayish-yellow. In his twenty-seventh case he speaks of a greenish-yellow mass within the softening. In the observation of Faurès the softening is called purulent.

In these six cases there is nothing to exclude the supposition that a syphilitic neoplasm may have been concerned therein. Even into the ganglia such a growth may intrude from the periphery, as from the substantia perforata or from the convolutions of the base.

Then, in the four cases where the softening was seated on the surface, a thickening of the soft membranes and adhesion to the cerebral cortex are mentioned—*i. e.*, in the cases of Gjör, Steenberg, and Zambaco; and the same remark holds good here which was made in regard to chronic meningitis. It may be distinctly affirmed that, with any examination less careful than should be demanded by the general modern development of cerebral anatomy, the possibility is always present that a small, yellow or grayish-red tumor may be overlooked at the bottom of a sulcus. I know from my own experience how often such a tumor is found in a softened spot, almost accidentally, after the autopsy is completed. This objection may be made to the observations of Gubian and Engelstedt.

The principal observations remaining are those of softenings or inflammations in the great ganglia, reported by Lancereaux, Steenberg, Ljunggren, and Oedmanson, which are much more like ordinary cerebral softening, and occur in a region where the development of a syphiloma belongs, to say the least, among the very rare cases. Here we must recollect what has been said above of arterial syphilis, and that softening of the great ganglia is the most frequent consequence of the disease of the vessels. This, however, was in former times usually overlooked, and thus the essential cause of the cerebral lesion failed to be recognized. In the five cases cited above the condition of the vessels is only once mentioned, and the meaning of the so-called inflammation in the ganglia must therefore be looked upon as doubtful. Only in Ljunggren's observation is it expressly said that the corresponding artery of the Sylvian fossa was healthy. But this case was evidently a real cerebral hemorrhage, and not an inflammation at all, and hence belongs in another category.

The fact deserves prominent mention at this place, that finding an actual cerebral hemorrhage, from the rupture of vessels, is as rare among the syphilitic as the occurrence of symptomatic apoplexies is common. Hemorrhagic infarction, from obstruction of vessels, is frequent enough; but the case just mentioned is the only one of genuine hemorrhage which I have found.

Finally, as to the three cases in which a general softness of the brain is mentioned, it is very questionable whether they were actually cerebral softenings, and not more appropriately to be included in the category about to be treated, where a decisive post-mortem lesion was generally wanting; for the phrases, general swelling and softening of the brain (Dufour), softening of the left hemisphere (Steenberg), tawny discoloration of the cortex (Mildner), point rather to a change in the amount of blood or lymph contained in the brain, such as may happen in the most various diseases, than to actual inflammatory or degenerative processes.

I think, therefore, that this conclusion, at least, may be drawn from the summary just given—and there is no case known to me in this category where any more accurate anatomical data are given—that the cases so far reported are not sufficient to justify the assumption of a simple inflammation within the cranium in syphilitic cases, neither originated nor accompanied by a new-formation.

The possibility of such an occurrence is not denied; but for the present the question is an open one, and any new cases of this kind should be most carefully examined as to any small tumor which may possibly be present, the condition of the vessels, and the histological character of the softened portion itself.

c. *Absence of all gross anatomical lesions*, after previous severe brain trouble in the syphilitic, has been stated by many writers as a condition often observed.

I have collected eleven observations of this kind, where somewhat accurate data are given—one each from Delaunay,[1] Gjör,[2] Engelstedt,[3] Ricord,[4] Kussmaul;[5] two from Zambaco;[6] four from Steenberg.[7] In all these cases the brain is described as completely normal, or something is said of hyperæmia or œdema, conditions met with in other cases by the hundred. The spinal cord also, when it was examined, offered not the slightest abnormality. In Kussmaul's case even the most careful microscopic examination (in how many places?) could not show anything abnormal. Yet in all these cases important nervous symptoms had been present, and had finally caused death.

If we examine the symptoms observed in these cases during life, we find that they may all be divided into two series: 1st, mental disturbances—either acute delirium, stupefaction, somnolence, or chronic disturbance of the intellect, with loss of memory and gradual dementia, sometimes preceded by delusions of magnificence (*délire des grandeurs; Grössenwahn*); and, 2d, paralyses, partly of the extremities, partly of the cerebral nerves, and partly of speech, or of the sphincters, not, however, making their appearance as distinct hemiplegias, but rather occurring on both sides, ascending, changing from one side to the other, coming quickly, but disappearing again, and, finally, as a rule, ending in general weakness—in short, behaving just as we observe these to do in dementia paralytica.

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[1] Leon Gros and Lancereaux, p. 173.

[2] Norsk Magazin. Bd. XI.

[3] l. c., Obs. 2.

[4] Leon Gros and Lancereaux, l. c., Obs. 78.

[5] Mercurialismus. 1861, p. 369.

[6] l. c., Obs. 71 and 73.

[7] l. c., Obs. 1, 2, 22, 34.

In general, of all the groups of symptoms occurring in cerebral syphilis, that of which we are speaking is, as we shall soon see, the only one which resembles dementia paralytica. It should be stated here, in reference to the progress of the disease, that the cases may be clearly divided into two groups, a rapidly and a very slowly progressive. In six of eleven cases the disease lasted from two to eight weeks; in one five, in another eight months; on the other hand, in one five years, in another twelve years, and in Schüle's case, soon to be described, twenty years. These rapidly progressive cases are just the ones which generally occur at an unusually early period of the syphilitic disease; in three of those above mentioned the nervous disease began within the first year after infection.

From the preceding short analysis of several observations bearing on this point, it may be seen that these cases, in which up to the present time no change of the nervous centres has usually been demonstrated, hold also an entirely peculiar position in regard to the symptoms, and are, in fact, the very cases exhibiting analogies with dementia paralytica, to which Hildenbrand, Steenberg, and others, with perhaps too strong a tendency to generalizations, have expressly referred. One can easily recollect how long the results of anatomical investigations in regard to the general paralysis of the insane remained completely negative, before the modern methods of histological study had overcome the previous difficulties. It may be hoped that in our cases, also, future inquiry will give sufficient anatomical explanations of the peculiar appearances during life.

A beginning has already been made in a case of Schüle (l. c.), where a less practised observer, especially of an earlier period, would perhaps have found only a few abnormities worthy of mention.

The case was that of an officer, aged fifty-two, who was under observation in an insane asylum for twenty years, and had presented the symptoms of dementia paralytica, as well as frequent outbreaks of confirmed constitutional syphilis. At the autopsy were found, besides a circumscribed gummous inflammation between cranium and dura mater, a hemorrhagic pachymeningitis, an old opacity and thickening of the soft membranes, and an atheromatous degeneration of the large arteries of the base, a peculiar pale gray, as it were swollen, condition of the cerebral cortex, a small softening of the left nucleus lenticularis, and a gray degeneration of the lateral columns of the spinal cord, chiefly on the left side. Upon microscopic examination, it appeared that in the cerebral cortex the texture of the neuroglia had taken on another and homogeneous quality, and was abnormally filled up with nuclei, single and in groups, chiefly along the vessels, which were themselves much altered. Their walls were thickened, sclerosed, and their cells had undergone fatty degeneration, or the channels were accompanied by close rows of nuclei, or by lines of spindle cells, others being surrounded by a dense web of connective tissue, or obliterated so as to become fibrous bands. The ganglion cells were shrunken in various degrees. (The other data in this very complete history are omitted, for the sake of brevity.) Here, then, in the cortex of this brain a profound and unusual lesion was found, which was only made manifest by the more delicate methods of investigation.

Symptoms.

It can easily be understood that with the great variety of anatomical appearances found in cerebral syphilis,[1] the symptoms in individual cases must also present very great differences.

Far asunder, however, as they may be in their progress and termination, at the beginning, or rather in their prodromata, they are all very much alike. The most frequent and striking symptom met with at this time is *headache.* It is almost never absent; the patients, who have any recollection at all, usually recall this as the first sign of disease.

It precedes the more distinctive appearances by days, or more frequently by weeks and months, or even years, and is regarded by many patients as something which has nothing to do with the later disease. It varies in severity, and often becomes almost insupportable, but is, like all pains, not always persistent to the same degree, occurring in paroxysms which, like the osteocopic pains of the extremities, become worse at night, often at about the same hour, and remit towards morning.

Long intervals may occur in the presence of this symptom; even without treatment the pain sometimes wholly disappears, to break out again with the greatest intensity a few weeks or months later. The seat of the pain is just as various. The whole head is seldom affected: but generally a lateral, anterior, or posterior half, or only one spot from which it radiates; or even a perfectly circumscribed region, as in clavus, which, however, on account of the great intensity of the pain, produces the same effect upon the general condition as general cephalæa. It is a highly important fact, not always sufficiently noticed, that this headache, when it can be localized, is generally made distinctly worse by pressure at certain points.

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[1] This designation is used for the sake of brevity. Syphilis of the membranes, arteries, etc., is included.

We believe that this symptom, so exceedingly common, must be looked upon as prodromal in the true sense of the word, inasmuch as in the majority of cases it is not dependent upon processes taking place within the cranium, but upon the outside.

If we recollect that in almost every case of cerebral syphilis signs of the constitutional disease are found also on the cranium in the form of deep or shallow cicatrices, osteophytes or gummous inflammations, that even during life gummata upon the cranium are observed to precede internal syphilis; that the patients often point to exactly the position of a cicatrix as the seat of the greatest pain; if we consider how very sensitive is the periosteum, while the greatest part of the brain substance is not at all, and the soft membranes only doubtfully so; and that, of the intracranial organs, the dura mater alone can be considered capable of intense pain, and that it (the dura) often remains unaffected; and if we reflect how long headache entirely alone may precede cerebral symptoms; and that the pain, if it originated at a point within the skull, could not be increased on pressure—we can hardly refuse our assent to this view.

In those cases where the dura mater is involved, the intracranial disease also may be accountable for the long duration of the pain as well as for a definite localization; but in this case the pain is no longer prodromal, but accompanies permanently the other symptoms, and is present under similar circumstances.

Besides the headache, a permanent or often recurring *sleeplessness* is found.

Often it is a consequence of the pain, which is much worse at night; but at other times it occurs in the intervals, and is then without apparent cause. Without any special disorder, an inability to sleep—which is particularly remarkable at the youthful age, at which so many syphilitic patients are found—makes its appearance, and often proves intractable to artificial aids. This phenomenon, too, may last long and lead to no little exhaustion. The direct cause cannot as yet be stated.

While these two symptoms, or at least one of them, are genuine prodromata, and in many cases very *remote* ones, the others, which have been called prodromal, and which might perhaps be called *nearer*, are in reality due to the developing stage of the disease itself. Among them belong the dizziness, which usually occurs in distinct attacks, and may even increase to fainting; the feeling of numbness in the head; mental disorder and confusion; shooting pains in the extremities; general discomfort; slight loss of memory; greater slowness of thought or speech, or a greater excitability of manner; unusual irritability of character, etc. All these symptoms arise, in one case or another, as the first indications of more severe disease, but are either too slight or too intermittent to seriously arouse the attention or anxiety of the patient (they often happen to healthy persons under the influence of varying frames of mind, etc.), and thus are not considered as a disease. Only when the patient is asked, in the midst of a severe attack, about his antecedents, are these trifling signs recollected.

These symptoms are manifestly dependent upon changes in the amount of blood or lymph contained in the brain, and upon other temporary processes accompanying the development of the new-growth. Lancereaux therefore calls them, perhaps with only partial justice, congestive. At any rate, let their seat be what it will, and no matter how various the future progress, they belong to nearly all classes of cases, and are therefore to be referred to general processes accompanying the special lesion in the interior of the cranium.

The disease itself makes its appearance with the most rapid and unexpected outbreak of severe cerebral symptoms. The nature of this outbreak, as well as of the further progress of the disease, is determined by the anatomical form in which the disease develops within the cranium. However various the course of the brain affection in individual cases, however manifold the expressions of the disease, certain fundamental types can yet be recognized in the majority of cases, and the cause of these typical distinctions between the several modes of progress can be found in their anatomical relations. The following sketch of the symptoms is undertaken with this object in view:

1. *Psychical disturbances, with epilepsy, incomplete paralyses (seldom of the cranial nerves), and a final comatose condition, usually of short duration.*

In this class of cases the severer group of symptoms, having been preceded for a longer or shorter time by the premonitions described above, often makes its appearance in the midst of apparent health, in the most sudden and alarming manner, by an epileptic fit, which is soon followed by more or less similar attacks, at first with completely healthy intervals, but afterwards with disturbances, especially of the psychical functions. At other times the early attacks are separated by a long interval; weeks elapse after the first catastrophe, and the patient begins to forget it. Then comes a new attack, and the development of psychical symptoms follows slowly. The latter may, however, even precede the epileptic attack. The epileptic fits are either completely developed, with loss of consciousness, fixed pupil, etc., or, as Wilks states, consciousness may not be completely lost, or rudimentary attacks (especially of epileptic vertigo) alternate with those which are completely developed. Sometimes the epilepsy remains for a long time as the sole symptom.

The mental disturbances consist at first in a somewhat bad temper or irritability, which, after a varying interval, passes on the one hand into melancholy, on the other into mania, and is sometimes rapidly followed by a marked weakness of intellect and memory. These last symptoms have some similarity to those which are observed in dementia paralytica; the great fickleness of disposition, weeping and laughing in the same minute, the fluctuation and want of fixedness of all the ideas, the slowness of thought, specially distinguish these mental disturbances of syphilis. According to Wille, however, the delirium of magnificence, so common in dementia, is wanting.

Certain paralyses now usually make their appearance, which are characterized, in this form of cerebral syphilis, by their gradual development. They do not occur in the form of an apoplectic attack, nor do they attain the completeness of paralyses originating in apoplexy, but appear rather as pareses or like conditions of considerable weakness. They are, however, usually predominant upon one side. The speech is peculiarly prone to suffer, and the modifications of the troubles affecting this function are very various. Pure and complete aphasia is not one of the most frequent. I have, however, seen it at least temporarily, for instance, during the increase of psychical symptoms, and there have been cases of persistent aphasia combined with agraphia. A peculiar hindrance to speech, for which the French have the appropriate expression, "embarras de parole," is more frequent. The patients speak either with remarkable slowness, or as if a leaden weight lay upon the thoughts; or they stammer and hesitate, as if in the middle of the sentence they had long forgotten what they wished to say at the beginning; or they make sympathetic movements of various kinds, as if the work of speaking called for an enormous expenditure of will power. The articulation, however, with all this, may remain completely unimpeded, the pronunciation of the single consonants, or the repetition of words pronounced before them, perfectly good, and the intellect (if not previously affected) clear, but the possibility of rapidly carrying out the impulse of the will seems to be wanting. Such patients are generally not able to read, or the first lines may be uttered fluently enough, soon to be interrupted by an increasing stammering; in counting, they cannot get beyond a few figures, etc.

This incapacity for speech varies much from time to time, and the patient can often, when unconstrained, succeed in doing what he is no longer able to do in society or before the examining physician. This all proves that we have to do with a disturbance seated beyond the prime motor centres, a weakness of those organs from which the stimulus proceeds to set these centres in motion—organs, therefore, which we must locate in the gray substance of the cerebrum.

Other paralyses are added, among them usually ordinary unilateral weakness, which is recognized by a more sluggish reaction of the corresponding limb, and gradually increases to a quite well-marked impediment to its use. In these cases a complete paralysis is rare. The paresis attacks sometimes only the upper, sometimes the lower, sometimes both extremities of the same side, and unilateral facial paralysis is also observed. A slight drooping of the shoulder, a dragging of the foot, a diminished energy of particular movements may be observed, without the power of walking, raising the arm, writing, etc., being completely lost.

These conditions progress with singular fluctuations; often increased energy returns for a considerable time, while the psychical disturbances described undergo a remission, and the epilepsy makes longer pauses. But finally, especially if no suitable treatment has been instituted, and often, in spite of it, an increase of all the symptoms takes place. In particular the epileptic attacks become more frequent, and may at last follow each other without cessation; in the intervals the intelligence is more and more disturbed, and a condition of somnolence, and finally of deep unconsciousness, sets in, with a general weakness of all the limbs, twitching of the extremities, and involuntary passage of the urine and stools. Fever, affections of the lungs, and bed-sores make their appearance, and the patient dies in deep coma.

With suitable vigorous treatment, this combination of disturbed intelligence, epilepsy, and slight paralysis—among which the convulsions usually attract the most attention—may be temporarily, or even permanently, cured, as many cases of recovery, especially from syphilitic epilepsy, prove.

This course of syphilitic cerebral disease usually depends upon those forms of anatomical change where some point on the convexity of the cortical substance is the centre of development of the new-growth—where, at some circumscribed locality in the subarachnoid space, a gumma is developed which leads, in the manner above described, to adhesions of the membranes, attacks the brain, and in its further extension produces softening of the cortical and adjacent medullary substance—or where a diffused gummous meningitis, attacking one or both hemispheres, is added.

It can easily be seen that psychical disorders in particular must be developed under the influence of a process seated thus at the periphery. Jaksch, who described this affection purely from a symptomatic point of view, mentions that among fourteen autopsies of patients mentally affected, in twelve the membranes and cortex were specially affected.

It can further be understood why, in these cases, strongly marked paralyses are usually absent, since the motor ganglia are not directly affected by the new-growth, but suffer secondarily by the increase of the quantity of blood in the corresponding hemisphere, the tension, etc. One can also easily see that cases of this kind may assume such a form that combination with well-marked paralyses must take place. It makes a difference whether the affection is seated in the posterior or anterior part of the brain, since in the latter the presence of special motor organs must now be looked upon as well established. Complete aphasia will be present if the lesion is situated exactly in the third frontal convolution; single cerebral nerves can be paralyzed (and such cases occur) when the affection is so situated that it both affects the cortex extensively, and also attacks portions of the base (for instance, in the neighborhood of the inner part of the base of the anterior and middle lobes). The most frequent paralysis of the cerebral nerves, which is found with the group of symptoms just described, is disturbance of vision, to the extent of blindness on one or both sides (according to Jaksch, in forty-five cases fifteen times); this, however, is sometimes the result, in such cases, of optic neuritis in connection with dropsy of the sheath of the optic nerve—an event due to leptomeningitis or to increased pressure within the cranium, without the optic nerve being directly involved by the new-growth. Such a case is described, for instance, by Oedmanson.

One point ought to be made particularly prominent, although its explanation with our present knowledge is not altogether clear, viz., that the anatomical form, now under consideration, is precisely that which is specially distinguished by the occurrence of epilepsy.

In twenty-six of my forty-five cases of new-growth in the cranial cavity, where the conditions were as described (the large tumors of the dura mater alone being also included), epileptic or similar convulsive attacks were observed twenty times; in the other nineteen, where the neoplasm was limited to the white substance of the base or to the middle of the brain, this symptom was found but twice. Jaksch found, in twelve autopsies of former epileptics, twice a gelatinous mass in the subarachnoid space, four times adhesion of all the membranes over a limited space, and once gummous nodules in the membranes, which pressed upon the brain. Hence the epilepsy of syphilitics is, in most cases, the consequence of an irritation on the surface of the gray substance of the brain, and forms an appropriate member of the train of symptoms described.

2. *Genuine apoplectic attacks, with succeeding hemiplegia, in connection with peculiar somnolent conditions, occurring in often repeated episodes; frequently phenomena of unilateral irritation, and generally at the same time paralyses of the cerebral nerves.*

This second form of syphilitic cerebral disease is not less frequent than the first, and is that which, from the great change in the symptoms during its course, and from the great variety of the accidents, specially deserves that character of capriciousness which impressed even the older observers as belonging to cerebral syphilis.

The prodromata are the same as in the former groups: headache, change of disposition, a melancholy, shy disposition, precede for a long time the more decisive outbreak. The beginning of the more serious affection shows itself in various ways. Sometimes the patient remarks, when feeling otherwise well, a sudden paralysis of some cerebral nerve, such as a drooping of the eyelid, an unexplained squinting, double vision, or dimness of vision, or loss of feeling in some part of the face. These paralyses may be preceded by irritative phenomena, of longer or shorter duration, such as spasmodic contractions in single muscles (facialis, abducens, etc.), or especially by neuralgias of the trigeminus, which are to be distinguished from the earlier headache by extending over definite regions, corresponding to branches of the nerve, and by the pains not being increased by pressure, or only at certain points (points douloureux). These strictly limited symptoms may last a long time, even a year, before anything follows; but usually more general phenomena are added not long after.

In other cases an apoplectic attack is the first symptom which, unexpected and alarming—just like the epileptic fit in the form described above,—comes to interrupt apparent health. Not unfrequently some exciting cause gives the impulse, for instance, excessive bodily or mental exertion, a long stay in a crowded room, excess in Baccho vel in Venere. Soon after any injurious influence of this kind, the patient suddenly loses his strength, falls, and remains, usually only for a short time, without consciousness; or his condition is such that, as if half asleep, he barely remarks what is going on around him, but is incapable of all movement; or he has a brief attack of dizziness, during which he has time to secure himself against falling, by assuming a safer position. When the patient somewhat regains his strength, he finds that another trouble has accompanied the attack, viz., a paralysis, and usually a hemiplegia; the arm cannot be moved, or walking is difficult, the speech badly articulated, and aphasia is often present, or may even, under some circumstances, be the only paralytic symptom. This condition—the impossibility of translating his clear thoughts into words—produces greater anxiety and excitement than the paralysis of a limb.

Finally, it may happen that the hemiplegia—in a manner somewhat analogous to the paralysis of the cranial nerves, just described—sets in when the sensorium is perfectly clear, so that, as happened in a case described by me, the patient can follow, step by step, the progress of a paralysis in a limb.

The further progress of the case may at first resemble that of an ordinary cerebral hemorrhage or embolism. The general health is speedily re-established, and the paralysis remains as a slowly disappearing residuum of the shock. Much more frequently, however, the disease is not at an end even for the time, but goes on to further and more serious disturbances of the cerebral functions. These latter—the somnolent conditions, soon to be more carefully described—may, like the paralyses of cranial nerves, the hemiplegia, the aphasia, etc., introduce the general disease, and only secondarily be followed by paralyses.

These somnolent conditions develop with comparative rapidity. The patients complain for some days or hours of increasing headache, or only of an unusual dullness and confusion, or great exhaustion and disinclination to work. A peculiar behavior may be noticed; it is evident that they are often completely absent-minded; they stare, without any special reason, at what is around them with a fixed gaze, etc. Now comes, in the midst of their work, an attack of loss of consciousness as a precursor, or the mental functions are so changed in the course of a few hours, not unfrequently during the night, that from this time the presence of a severe cerebral disease is evident, even to the laity. The condition which has now established itself has in its whole aspect something very peculiar—I might almost say characteristic of cerebral syphilis. The train of symptoms stands about midway between the two descriptions, which are usually given, of meningitis and circumscribed softening of the brain. It has some analogy with the hydrocephaloid disease of children, as described by Marshall Hall.

The patients are found in a typhoid, half conscious, half sleeping condition, like drunkenness, from which they can be only very temporarily awakened, but which is frequently combined with a purposeless instinctive activity, which has not the character of simple automatic movements, like that of ordinary meningitis, but rather presupposes a sort of combination of half unconscious motor impulses. They usually lie in bed, with closed eyes, with a certain fretful, disturbed, though sometimes cheerful expression of countenance, the forehead wrinkled, or the mouth as if voluntarily distorted. Thus they may lie, if unmolested, as if half asleep, for a great part of the day; but occasionally there are times, especially towards evening, when, of their own accord, they become restless, and, if not paralyzed, get out of bed, call for their clothes, or get into another bed; married men ask after their wives, etc. Often a special disposition is noticed to have the hands upon the genitals, to play with them, and constant erections may take place. Frequently they pass stools and urine in bed or in the room—not, however, in consequence of an incontinence of the sphincters, but of erroneous ideas: the whole behavior generally showing that the patients think they are in the street, upon the night-stool, etc. All these actions are carried on in the same half-sleeping—I might say somnambulant condition, in which they had previously lain in bed. In the midst of them they may be waked up, carried back to bed, and then remain quiet for a time. If one approaches them as they lie, apparently sleeping, in bed, it will be noticed that they are not completely insensible. After they have been called, shaken, etc., they open their eyes, astonished, and gaping and sighing, like healthy persons just awakened, give to the questions asked them hesitating, stammering answers, which are often, as appears by comparison, perfectly correct, but frequently betray a great diminution of mental activity, especially of the memory. Then they immediately sink back in sleep, often groaning, and complaining of the head. If an attempt is made to examine them, sometimes they permit it to be done quietly, at others they defend themselves with hands and feet; while sometimes there are only certain procedures which they obstinately resist. It follows from these symptoms that we have to do, not with complete abolition, but with a more or less severe injury to the higher mental functions.

According to the nature of the case, in other particulars, and according to its method of progress, we find upon examination some one of the motor paralyses described above—for instance, among the anomalous conditions of the cranial nerves, frequently deviations in the action of the muscles of the eye, and in the extremities more or less decided hemiplegia. When this is present, it is often connected with contractures or unilateral clonic spasms (actual epilepsy being absent); while frequently a certain muscular stiffness in that limb, which the patient can move in every direction, or a tonic contraction of certain muscular groups, is all that can be noticed. Sensibility (as Lancereaux has already mentioned) is generally much less or not at all disturbed; the prick of a pin is everywhere perceived, as the motions of the countenance or other parts of the body show, and reflex movements occur. I have occasionally noticed in such cases even a peculiar increase of reflex irritability upon one (the contractured) side, so that by a slight touch one could excite intense spasm of the whole body. Cases often occur where peripheral paralyses are completely wanting, and the somnolent condition alone attracts attention. Upon examining the whole body, sometimes no further disclosures are made. Usually, but not always, one finds some recent or ancient trace of syphilis—exanthem, gumma of the skin or bones, syphilitic testicle, general swelling of the glands; or sometimes, upon more minute investigation, remains of an older lesion upon the penis, etc., may be demonstrated.

The other internal organs show no change; the bowels are somewhat confined, the patients eat what is given to them, or occasionally nourishment is refused. Sometimes, but not constantly, moderate or even very high fever is present.

This condition lasts with only a few interruptions (often comparatively quiet or almost perfectly clear intervals) in a few cases for hours, in most others days and even weeks. It may pass in a short time into a constantly heavier and deeper sopor, and finally into permanent coma, which leads immediately to a fatal result. Thus, in fact, the earliest appearance of this group of symptoms may already introduce the fatal catastrophe. But what gives to this disease its special peculiarity, is not the somewhat rare occurrence of a rapid transition into symptoms leading to a fatal end, but rather the fact that this cerebral disturbance, apparently so severe, and hardly capable of recovery, may sometimes, under suitable treatment, but sometimes without it, be completely removed, and again give place to an almost normal condition. The peculiarity which, in addition to the great variety of the individual symptoms, gives to cerebral syphilis this paradoxical, vacillating, startling character, is this, that its accidents, just as unforeseen they developed up to a certain intensity, may again vanish in the same remarkable way.

In the condition which we have before us, this improvement takes place gradually—the intervals between the exacerbations becoming longer and more complete; but often, after the patient is supposed to have recovered, comes another slight relapse of confusion, delirium, etc. Finally (and in general when the severe symptoms have lasted some three or four weeks, about the same time is necessary for recovery) the patient may again become completely restored to activity, although a careful eye notices that he has received a shock, that his mental capacity is in some respect deficient, that the speech has not its former fluency, the memory remains affected, etc. For one, however, who had no previous acquaintance with him, the patient appears, under ordinary circumstances, perfectly well.

In a similar way improvement of the motor paralyses takes place, although indeed the hemiplegias do not in general recover with the same rapidity as the symptoms just spoken of. The changes of the cerebral substance by which, as we shall see, they are produced, are much too deeply seated. We therefore ordinarily observe that in such patients, who are in general paralyzed during this severe cerebral disease, the traces of the paralyses remain long after the acute affection has disappeared: the gait is dragging, or walking is only possible with the help of a cane; the arm is partly or wholly useless, its muscles not unfrequently in a condition of slight contracture, and even a moderate facial paralysis or defects of articulation may last for a considerable time.

Yet, after months and years, even these severe hemiplegias, if no new attacks intervene, may slowly but completely disappear, and the patient again become in all respects approximately sound.

Sometimes, in addition to the form of paralysis just described, there also occur in this kind of cerebral syphilis other hemiplegias, which have a much more transient character. They do not usually arrive at the same degree of development as the genuine hemiplegias, but yet at the beginning of the severe cerebral trouble, when consciousness is lost, an arm, a leg, half of the face, or all one side of the body may become useless, and the situation appear at first quite similar to that observed in the severe hemiplegias. But on the next day, or some days later, this weakness begins to grow less, and, to the astonishment of physician and patient, every appearance has vanished within from five to eight days. This may even happen, after a previous apoplectic shock, without succeeding loss of consciousness; and also the rapid improvement of the paralysis may coincide with the continuance of the somnolent condition. Each is independent of the other.

The paralyses of cerebral nerves—the third series of symptoms—of which the second form of cerebral syphilis is composed, may behave in the same way as the hemiplegias. Here also we often find entirely unexpected improvement; but in general the type of protracted recovery is the prevailing one, as it also is in the hemiplegias.

Thus we can here see a multitude of morbid phenomena recover at various intervals—in part, after a short existence, and in a most unusual way.

Yet it is here very much as it is with the external syphilides. If energetic treatment has not been instituted, the apparently perfect health is not to be trusted. It is not only that slight remains of the disease, as above mentioned, still peep out, but at every occasion a new outbreak is threatened, and frequently enough occurs. Then the scene sketched above is repeated, with sometimes one and sometimes another modification in the details, such as a new paralysis on the other side of the body, or the affection of another cerebral nerve.

But even now a kind of spontaneous cure can again take place, though some of the paralyses require a longer time for improvement. Often in the third or fourth attack no new paralysis appears, but merely the drunken-like condition, which may rapidly disappear again. We have here, therefore, a complete series of separate episodic diseases, with intervals of relative health, which, taken together, constitute the general mode of progress of this form of venereal cerebral affection. When energetic treatment is not instituted, the separate attacks come nearer and nearer together, the paralyses increase, the somnolent condition passes into final coma, which, in almost all forms of cerebral syphilis, precedes the fatal issue by days or weeks.

If the patients, after this long-enduring and manifold disease, come upon the autopsy table, a different state of things is found from that in the first form. If a new-growth is present, it is generally found at the base, covering more or less space, sometimes in the subarachnoid space, and sometimes proceeding from the dura mater, and projecting to a variable depth into the interior of the brain. The paralysis of the cranial nerves, which was present, always finds its explanation in the extent of the new-growth; yet the latter (especially in the form of gummous meningitis) may be present in such a shape that the nerves which it surrounds are not injured by it, in which case the paralysis must have been wanting during life. But with very special frequency is found—in addition to the new-growth in the membranes, and in extent entirely independent of it—the syphilitic disease of the arteries described above. Almost all the observations of this kind collected by me ran their course according to the type just described.

Finally, while spots of softening are usually wanting in the cerebral cortex, and in the large mass of medullary white substance (centrum ovale), we regularly find red or yellow softenings or infarctions, cysts, etc., in one of the great ganglia, most frequently in the nucleus lenticularis and nucleus caudatus, more rarely in the thalamus, corpora quadrigemina, pineal gland, etc.

It is evident that while the paralyses of the cranial nerves, in this form of the disease, are produced by the exudation at the base of the brain, the hemiplegias are due to the changes in the great ganglia. Not unfrequently after such changes have lasted for a long time, the most beautiful secondary degenerations can be found extending from the crus cerebri to the lowest portion of the spinal cord; and, from numerous pathological observations, there can no longer be any doubt that the anatomical changes in the corpus striatum interrupt voluntary motor activity more completely than can any softening at any other part of the brain.

The softenings or infarctions of the motor ganglia which are here found may always, in the presence of vascular degeneration, very naturally be brought into connection with the specific disease at the base, and are not of themselves specifically syphilitic affections, but equivalent to the lesions which may occur in connection with thromboses or embolisms of any kind affecting the arteries at the base. In the anatomical description of the arterial disease we have seen that we have to do with a proliferation, starting from the inner coat, which, in a short time, greatly narrows the lumen, opposes material hindrance to the current of blood, and may either produce by itself complete obliteration or give rise to the formation of a clot which shall plug the vessel. The development of the vascular degeneration and the consequent disposition to thrombosis always takes place earliest and most intensely in the large vessels which go to form the circle of Willis, in the circle itself, and in the main trunks of the six large cerebral arteries originating from it (arteriæ corporis callosi, arteriæ fossæ Sylvii, arteriæ cerebri profundæ vel posteriores). In order that the effect of a closure of the vessels taking place at this point may be understood, we must previously describe the arrangement of the cerebral arteries. This is of such a character that the gray cortical substance of the brain, and the white medullary substance connected with it, as far as the roof of the ventricles, is supplied with blood-vessels on an entirely different plan from that which exists in the white substance at the base, and in the large ganglia which lie above it and form the floor of the ventricle.[1] So far as the arteries lie upon the white substance—that is, so far as they consist of such great vessels as the vertebrals, basilar arteries, carotids, circle of Willis, and some two centimetres at the origin of the anterior, middle, and posterior cerebral arteries (basal region)—they give off small arteries of less than one-half mm. in diameter, in a perpendicular direction, which penetrate directly downwards into the nervous substance, there branch, and, as so-called terminal arteries, without receiving any collateral branches, break up in the ganglia into capillaries, and pass into the smaller veins. But from the point where they attain the surface of the gray substance they run for long distances in the pia, without giving any large arteries to the brain, but dividing in the pia, no longer in three but in two dimensions, break up into constantly smaller arteries, which mutually distribute numerous collaterals, and thus form a network by which not only the several branchlets of a main branch, but the various main arteries are put in connection with each other. (Thus, in this region, the collateral arterial system prevails; in the former, the terminal.) Then from the superficially spread network of the pia very small, already capillary vessels penetrate directly into the brain substance (cortical region).

If we now imagine the syphilitic process in the arteries developed to such an extent that, in a comparatively short time, a closure of the lumen at a circumscribed portion takes place from coagulation of the blood, it is evident that those regions will be most imperilled where the terminal arteries (*Endarterien*) penetrate directly into the brain—that is, the basal region. For, by an obliteration at such a place, the circulation in the small lateral branches of the main trunks, each forming for itself a terminal artery without anastomoses, and hence having no collateral assistance, must be stopped, and those processes must take place in them and the surrounding tissue which have been so thoroughly described by Cohnheim, and which express themselves in the brain as red, yellow, or white softening, according to the varying accessory phenomena. But since that section of the cerebral arteries, to which the large vessels of the base belong, is the most frequently and most severely affected by this disease, and since it is from these that the great cerebral ganglia receive their (terminal) arteries, it is evident that the so-called softenings must occur most frequently in these regions, and the explanation of the syphilitic hemiplegias above described becomes easy.

In the cortical region the stoppage of a large artery does not permanently exercise so marked an effect, because the peripheral portion of the plugged vessel can be indirectly provided with blood by the collaterals found in the pia. Nevertheless, at the moment of origin of a rapid occlusion, an effect may be manifest, for there is then for a short time a sudden stoppage of the blood stream, and hence a material diminution of tension in the corresponding parts of the brain, but afterwards, with the renewed supply of blood from the collaterals, as always happens in vessels the current of which has only been for a short time interrupted, an increase of pressure, even beyond the degree previously present, takes place.

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[1] Compare *Heubner*, Hirnarteriensyphilis, p. 170. et seq.

Thus a very considerable territory of the gray cortex is exposed to variation of pressure, transitory, to be sure—always under the entirely possible condition that the thrombosis shall take place rapidly. The transitory injury to a portion of the brain substance, which results from this change in the pressure, is able to evoke disturbance of its functions and symptoms of a transient character, particularly if we consider that with the activity of the collateral arteries in respect to the ischæmic region, a considerable embarrassment of the whole circulation of the hemisphere arises. In this way the temporary apoplectic fits may be explained which sometimes, but not always, accompany the onset of syphilitic hemiplegia.

Finally, a narrowing affecting a large number of arteries, even without total stoppage, especially since in our case the narrowing affects longer and longer portions of single vessels, must make an injurious impression upon the whole brain. The resistance to the current in tubes thus narrowed increases considerably, and proportionally to the length of the narrowed portion. Hence a useless consumption of the momentum of the blood current, the consequences of which are intensified by the fact that with the diminution of the elastic force in the arterial wall a subtraction takes place from the sum of the forces which tend to propel it. The blood, therefore, moves more slowly and with less tension in the network of the pia mater and in the brain capillaries. Oxygenation is diminished, and the nervous elements suffer a loss in their functional capacity. This effect, if the cause continues, can be no temporary one, and must show itself in symptoms of longer duration. Here, it seems to me, we have found the key to understanding the peculiar somnolent or drunken-like conditions in which the cerebral functions are not lost, but greatly interfered with.

The peculiar tendency of these week-long accidents to retrogression or even complete disappearance, notwithstanding the still present but not progressive disease of the arteries, may find its explanation in the possibility of the nervous substance gradually accommodating itself to a change of tension.

Thus, finally, the most important symptoms of this form of syphilitic cerebral disease are to be referred to the affection of the cerebral arteries.

3. *Course of the cerebral disease similar to that of dementia paralytica.*

The disease begins with a general uneasiness, feeling of discomfort, disturbance of the general health, melancholy, or symptoms of psychical irritation, unusual mental activity, transient confusion, and sometimes even—though much more rarely than happens in the ordinary dementia—with the monomania of magnificence (*des grandeurs*). These psychical disturbances last for a long time without somatic nerve troubles; they may disappear, and an apparently normal condition return. Then they appear anew; and thus a changeable condition may last for years. It is to be remarked here, however, that the acute exacerbations of the psychical affection are generally accompanied by a new outbreak of a syphilitic exanthem or some other manifestation of the constitutional disease in the bones, throat, nose, etc., which is not always the case in the forms of cerebral syphilis previously described. Mildner, l. c., and Wright[1] describe cases of this kind, and call special attention to the circumstance. Bodily symptoms now gradually make their appearance, at first consisting in a general weakness and loss of power. The patient becomes easily fatigued and is no longer capable of severe exertion; the movements become uncertain, and the gait staggering. Subjective disturbances of sensibility, formication, numbness in single limbs, and shooting pains appear, but all in very slow and gradual development. The speech is hesitating, stammering, uncoordinated; the tongue begins to tremble and make irregular movements; while speaking, memory, and intelligence decrease slowly but steadily. Now comes a period of actual paralyses, either hemiplegic or paraplegic, with or without loss of consciousness. They are, however, essentially distinct from the hemiplegias of the preceding form; they do not persist, but go as quickly as they came, often the same day or the day after; return after a short time, sometimes at the same, sometimes at another place, to yield just as rapidly a second time. So it happens that at some time, on lifting a weight, or from some psychical irritation, suddenly one arm fails to act, trembles, and remains immovable for some hours; at another time the opposite leg is suddenly paralyzed, so that the patient falls, but the next day is in a condition to walk; or both the lower extremities become for a short time paraplegic; or again the speech is absent, or stammering and indistinct, but in a little while improves, etc. In a case under my observation such transient pareses were repeated twice a week during four months, later came only every ten weeks, and finally ceased to appear, while the psychical functions and ability to speak became gradually less and less.

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[1] Edinb. Med. Journ. June, 1872.

During the period of these transient paralyses the general bodily weakness makes simultaneous steady progress; the patients become more and more incapable of any vigorous movement, going upstairs, etc. Upon combined movement, a well-marked ataxia makes its appearance; upon putting out the tongue uncoordinated accessory movements occur; the writing is irregular and finally illegible, and the gait staggering, dragging and ataxic. In the meantime the subjective disturbances of sensibility increase, and transient pareses of cerebral nerves and inequality of the pupils associate themselves thereto. Later, paralysis of sphincters occurs. The whole course extends over months and years, interrupted by long intervals of improvement, and after several years the patient dies from a cystitis, bed-sores, or some phthisical or acute affection of the lungs.

We have been obliged to make mention of the form of cerebral syphilis just described in the anatomical part of this treatise. It was the cases of this kind in which, when death took place early from complications, one was usually unable to recognize any abnormity in the central organs, or, when the disease had lasted for a long time, an apparently slightly characteristic opacity of the soft membranes and a little atrophy of the convolutions were observed, which seemed to furnish no satisfactory explanation of the symptoms during life. Whether repeated hyperæmias or other morbid processes lie at the bottom of the appearances—as Lancereaux assumes in his "Congestive Form of Syphilitic Disease of the Brain"—cannot be answered with certainty either in the affirmative or in the negative. The absence of any decisive appearances to be found in the body of a person dying in the early stages of the disease, argues nothing against the possibility of the presence of such vascular disturbances, especially if nothing but a macroscopic examination has been made. The non-venereal dementia paralytica is ranked, according to Meynert, as a consequence of often-repeated hyperæmias, which affect especially the gray cerebral cortex, and of this again the parts belonging to the anterior lobes. Only secondarily to this do the proliferative processes of the neuroglia leading to atrophy set in.[1] Macroscopically in the individual case hyperæmia is not likely to be very prominent at the autopsy, even while the histological examination demontrates the thickening and thrombosis of the small vessels, cellular proliferation of the adventitia, and accumulation of pigment flakes along the course of the capillaries.

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[1] Comp. *Lubimoff*, Virch. Arch. Bd. 57. 1873.

It may be mentioned, in this connection, that, according to Petrow, deep-seated disease of the sympathetic may take place in syphilis, and a degeneration—for instance, of the superior cervical ganglion—might certainly, under some circumstances, have an influence upon frequently recurring hyperæmias. In order to settle this question, the most minute attention should be given, in any future cases of this kind, to the microscopic examination of the cortex, and especially of the anterior part of the brain. I myself found, in the case of a man who died of phthisis, in the third year of the disease, which pursued a course somewhat like that above described, upon the examination of numerous portions of the cerebral cortex (especially of the anterior part, of the island (of Reil) among others, and particularly of both left central convolutions) around the (injected) capillaries of the cortex, an unusually marked proliferation of nuclei, while no increase of the neuroglia cells could be made out. I do not describe the case more particularly here, because only an examination of pieces of brain, hardened in alcohol, was possible: and other methods seem to me necessary for determining the more minute changes in the vessels. The case of Schüle, already mentioned above, certainly proves that we have to do in these cases with processes which take place around the vessels. Consequently, in all probability, minute processes in the cerebral cortex must be looked upon as underlying these so far mysterious cases, and not only the preceding mental disturbances, and the general weakness, but even those remarkable temporary paralyses need no longer be so incomprehensible, since we have obtained from Hitzig's investigations so much knowledge of the motor centres in the cerebral cortex. It is not worth while to attempt to go further, since we should be dealing only with hypotheses, until a careful description of more numerous cases of this kind is at our command.

As to the *duration* of syphilitic cerebral disease, this depends essentially upon the form in which it has appeared, and what treatment the patients have undergone. The second form, in which the vascular degenerations play the chief part, seems to run its course with comparatively the greatest activity. Here we may observe cases which, after long but undecisive prodromata, lead, within a few days, to death, in the shape of an apoplectic shock, with consequent coma, and where a multiple thrombosis of several important arteries is found. More frequently, however, even here the disease lasts a longer time, several weeks or months, and when an energetic anti-syphilitic treatment has been instituted the disease may be seen, even when a cure does not take place, to be protracted for several (up to four) years.

The first form does not lead to a fatal result so rapidly; the epilepsy, which is so frequent a companion to the other cerebral symptoms, may last for several months, before any serious disturbance of the consciousness or of the psychical functions sets in; and even after such disturbances have appeared, the severer brain disease may, even in insufficiently treated cases, still last for weeks and months before death arrives. Here we find, sometimes with and sometimes without treatment, fluctuations, inclination towards recovery, etc., which protract the disease even longer.

The third form has the longest course, as it may last many years; and, if it ends in a short time, within the first year, for example, a complication on the part of the lungs is generally responsible for the early close.

The *termination* of the disease may be, and in fact frequently enough is, a fatal one. The impairment of a great mass of brain substance, the destruction of nervous organs important for life, the peripheral affections, which, in connection with the brain disease, arise in the skin, the lungs, and the bladder, furnish a sufficient explanation for the fatal catastrophe which we so often meet.

But, on the other hand, when an efficient anti-syphilitic treatment is begun early enough, an absolute or relative cure of the disease, whether it appears in one form or in the other, is decidedly possible. We find among the records of cases no small number of epilepsies—even those which were connected with psychical disturbances—where a complete cure was obtained, and that too while the presence of a new-growth on the surface of the brain was to be supposed. We find cures recorded, further, in those cases, where, besides paroxysms of a general brain disease, well-marked, unilateral paralyses were present. Of course, if severe and lasting hemiplegia have once taken place, as after actual destruction or cicatrization in the substance of the brain, a complete removal of the paralysis can seldom be obtained, and the patient is only relatively cured. Finally, among the histories of cases, especially those published by Hildenbrand and Steenberg, we find some which correspond to the picture of the disease sketched under the third form, and where an energetic treatment produced a favorable result.

It must indeed be confessed that many cases which come under treatment after the affection has already lasted a long time—and indeed, some of more recent date—obstinately resist energetic anti-syphilitic treatment, and lead to death; in this respect the arterial diseases appear to belong among the most dangerous.

II. Syphilis of the Spinal Cord and its Membranes.

Pathological Anatomy.

The recorded cases of affections of the spinal marrow, produced by syphilis, are still few in number and incomplete, particularly with regard to the anatomical lesions, and consequently the description of these affections can only be fragmentary. However, after the preceding exposition of the various forms under which syphilis manifests itself in the brain, it is possible to study these cases by comparing them with the anatomical lesions found in that organ; when studied in this way, a striking analogy is revealed between the lesions of the two organs. It may be safely asserted, that lues venerea of the spinal marrow occurs far less frequently than syphilis of the brain. We do not propose to consider here the exostoses, periostites, etc., of the spinal column, which may affect indirectly the spinal marrow, as they must necessarily produce the same effects as do other affections of the bones, and will be described in the proper chapters. Aside from these, the following lesions have been observed in the spinal cord.

1. *Syphilitic neoplasms.*—Their favorite place of development appears to be at the *periphery* of the cord in the pia mater, in the subarachnoidal space, or on the inner surface of the dura mater and the layer of the arachnoid lying on it. Here, as in the brain, it leads to the agglutination of the three membranes and the surface of the spinal marrow, and seems partly to grow into the substance of the cord from without, and, partly by pressure on or destruction of the roots of the nerves arising from the part, to render them incapable of exercising their functions. The new-growth is not so apt to assume the form of a sharply defined tumor, as of a circumscribed infiltration of the membranes and their lymph-spaces with the newly-formed gummy material. This has been found both in the form of a fresh, reddish-gray, gelatinous new-formation, and in that of a yellow, dry,

caseous mass. Zambaco[1] reports the case of a man, thirty-five years of age, who, five years after the first infection, was seized with a most severe attack of external and osseous syphilis, which was followed by great general debility and anæmia, and by paraplegia of the lower extremities with partial anæsthesia and excentric pains. At the autopsy a gelatinous, gumma-like effusion was found encircling and compressing the cord in the lower part of the dorsal and the lumbar regions. Wilks[2] reports a case of the disease which commenced with rigidity of the dorsal muscles, anæsthesia and paralysis of one-half of the body, involved subsequently the other half, and terminated in complete paraplegia of the lower extremities. At the autopsy he found on the cord in the lumbar region an irregular oblong deposit (three-quarters of an inch long and composed of yellow, amorphous masses), which enveloped the posterior roots of the nerves, and was intimately connected with the cord itself. Yellow nodules and cicatrices were also found in the liver and the lungs.

In Wagner's treatise[3] a case is described in which a yellow nodule, as large as a small hazelnut, was found situated centrally within the cervical marrow. But as the autopsy revealed no other sign of syphilis, it is doubtful whether the case properly belongs here. I have been unable to find any other observation of syphiloma developed in the *interior* of the cord recorded in the literature of the subject.

Besides the two above-mentioned forms, syphilis seems also to occur in the form of very *small*, *multiple*, and *disseminated* new-formations on the membranes of the spinal cord—a form which might perhaps be termed miliary syphilis. To this form belongs the case observed by Engelstedt,[4] in which, in addition to similar new-formations upon the dura mater of the base of the skull and on the under surface of the tentorium cerebelli, a great number of closely grouped nodules of the size of a hempseed, tolerably consistent, and some of them easily separated from the surface on which they lay, were found symmetrically distributed on both sides along the entire length of the spinal cord upon the inner surface of the dura mater—*i. e.*, in the arachnoidea. Not long since a similar case was observed in the hospital at Leipsic.

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[1] l. c., Obs. 84.

[2] l. c., Obs. 4.

[3] Arch. d. Heilk. IV. 1863. S. 169.

[4] l. c., p. 145.

2. *The syphilitic callus.*—In syphilitic individuals who had during life presented symptoms of an affection of the spinal cord, a circumscribed induration of the cellular tissue from one to several lines in thickness, and starting either from the outer or from the inner surface of the dura mater, has been found in the vertebral canal. If it start from the outer surface, it leads to a close adhesion of the dura mater to the periosteum of a part of the vertebra, with consequent destruction of the vascular peridural adipose tissue; if from the inner surface, the result is a firm adhesion of the three membranes of the spinal cord, which can no longer be separated from one another. In the latter case the cord itself is involved in the process, and examination reveals over an area corresponding to the adhesions a proliferation of the neuroglia with destruction of the medullary sheaths of the white fibres, changes which may extend to a considerable depth. There can be no doubt about the specifically syphilitic nature of this neoplasm, which perhaps, from the very outset, was less rich in cells than the usual gummy tumor, and which, in the course of time, under the use of anti-syphilitic treatment, became transformed into such an indifferent callous mass of cellular tissue, that it presented at the autopsy nothing either macroscopically or microscopically characteristic of syphilis.

A case, belonging to the first variety, is reported by Virchow.[1] An army officer, of middle age, who had suffered much from syphilis, was seized with stiffness in the nape of the neck, and pains in the neck and arms, and finally with paralysis of both arms. At the autopsy, besides numerous syphilitic affections of the bones, the dura mater at the level of the fifth and sixth cervical vertebræ was found to be increased to three times its normal thickness, and bound down to the bodies of the vertebræ by a large amount of firm connective tissue.

A case, belonging to the second variety, was observed in the Medical Clinic at Leipsic. This case is so characteristic, and at the same time so unlike any of the cases that are described in the literature at my command, that a short description of it may be appropriate here.

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[1] Nat. d. const. syphil. Krankheiten. Archiv, Bd. XV.

St., aged thirty-seven, colporteur, exhibited, from his eighteenth to his twenty-second year, many symptoms of syphilis. In his thirtieth year he had pain in his left arm for weeks at a time; in his thirty-second year swelling of the left knee, enlargement of the spleen, and a cachectic appearance. In his thirty-third year, February, 1866, he was attacked with pain in the neck and in the right arm, and later on in the right leg and the left arm; in March, paræsthesiæ of the right arm, *without* loss of sensibility, supervened with rapidly increasing general debility. On his admission, March 6, there was complete loss of power in the right arm and partial loss of power in the right leg, whilst sensibility was unimpaired. During the month, without any specific treatment, there was some improvement of the paralysis, which, however, grew worse again in April. The left arm now began to grow weaker, and a few days later the left leg also. Spasms in all the extremities, paræsthesiæ (formication, etc.), but without the slightest diminution of the cutaneous sensibility, stillicidium urinæ, and severe pains in the neck were next developed, all in the course of one week. Towards the middle of April the paralysis became more complete. Difficulty of respiration set in—first the *inspiration*, then the *expiration* becoming more difficult; the articulation became indistinct; paresis of the tongue and ptosis of the right side were developed; deglutition became difficult, and the fæces were passed involuntarily. The patient hovered between life and death. At that time nothing positive was known about the former syphilis; but it was decided to try anti-syphilitic treatment as a dernier ressort.

Treatment by inunction was begun on April the 13th, and between that day and the 10th of May one hundred and twenty grammes (circa thirty drachms) of the milder mercurial ointment were rubbed in. Under this treatment a remarkable though gradual improvement took place. First, the patient was able to move his left arm, then the respiration became freer, and soon he could move the left leg; on April 22d he could move some muscles of the right arm, and on April 24th the toes of the right foot; on May 1st, he was able to pass his urine voluntarily. The pain in the neck, rigidity of the muscles, and spasms, however, still continued. Between the 18th and 30th of May thirty-two grammes (a little over eight drachms) more of the ointment were used by inunction. On June 11th he was able to walk for the first time; on June 26th he walked through the room without assistance; on July 6th he went upstairs; and on July 23d he went out of doors. A relapse set in in the beginning of August, and during the first half of September thirty-six grammes (about nine drachms) of the ointment were used by inunction. A discharge from the left ear now made its appearance; but otherwise the condition of the patient improved so much that he was discharged, on December 22d, with complete use of all his limbs.

In the beginning of February, 1867, the right leg again became heavier, so that he had to use a cane, but was still able to attend to his business. In May, 1867, he returned to the hospital, complaining again of weakness of the lower extremities.

He suffered also for several months from a scorbutic affection of the legs and from intestinal catarrh. In August improvement set in; he was able to walk around, but his gait was uncertain and he frequently fell.

In the beginning of 1868 a new relapse of the nervous affection set in, which followed exactly the course described above, and culminated (in April) in complete paralysis. The treatment by inunction was again adopted. About the middle of May, although there was but slight improvement in the paralysis, a careful examination revealed an almost perfect preservation of the senses of touch, of pressure, and of heat, and of the faculty of localization on the right, which was the more affected side, as well as on the left side. Only the combined muscular sense and sense of touch in the right hand was distinctly lessened; the patient, after repeatedly examining a small leather pocket-case, by handling it while his eyes were closed, thought it was a knife, etc.

In June an improvement in the paralysis set in. A tophus on the forehead was noticed, and a relapse of the scorbutus took place. A commencing infiltration of the superior lobe of the right lung was discovered.

In July the patient was again able to walk, but with well-marked *ataxia*. He was unable to walk or to stand with his eyes closed.

Discharged on the 15th of August; he remained tolerably well till September; but from the middle of that month the sensation of heaviness increased, and the paræsthesiæ and spasms of the right leg reappeared.

He entered the hospital the third time about the end of October. Under treatment with local faradisation the power of motion improved until the end of February, 1869.

The patient was again discharged, and for a long time the disease seemed to remain at a standstill. He followed his avocation as a colporteur during the years of 1869 and 1870; he was frequently seen on his rounds, and was observed to walk in a stooping posture, leaning on a cane, and with a marked ataxic gait.

He was attacked with a violent bronchitis in October, 1870; and on November 11th was readmitted to the hospital with symptoms of a febrile phthisical affection of the lungs, dyspnœa, night-sweats, etc. He died on the 18th of December, 1870.

At the autopsy, in addition to caseous infiltration of the upper lobes of both lungs, callosities of the pleura, etc., the following lesions were discovered in the central organs: On the bones of the cranium there were numerous excavations, and in a few places small perforations, and close by them thickening of the bone, and exostoses. Dura mater of the brain injected; accumulation of lymph in the subarachnoideal space; and numerous opaque spots in the soft membranes. Cortical and medullary substance hyperæmic, but otherwise normal. Corpora striata, thalami optici, corpora quadrigemina, cerebral peduncles, pons Varolii and the lateral ventricles healthy.

At the commencement of the right arteria fossæ Sylvii, a slight thickening of its wall was observed. The ependyma on the floor of the fourth ventricle, corresponding to the upper part of the medulla oblongata, was markedly thickened and its blood-vessels greatly injected. A transverse section of the upper half of the medulla presented nothing abnormal; but a section, made immediately above the opening of the central canal, revealed a gelatinous mass, which extended from the floor of the ventricle to the depth of two mm. (one-twelfth inch) into the medulla. In the region of the decussation of the pyramids the medulla was slightly constricted; and in this situation, corresponding to the left substantia reticularis, a transverse section revealed a softened condition. The pia mater and arachnoid were remarkably dark in this situation. About one ctm. (two-fifths inch) lower down, the dura mater spinalis was firmly adherent to the atlas on one side, and the pia mater on the other, and was about four mm. (one-sixth inch) in thickness. This adhesion extended over the posterior surface of the upper part of the cervical medulla to the extent of one ctm. square (two-fifths inch square). In this place the posterior pyramids were very hard and transparent. There was no distinct line of demarcation between the gray and white substances in this part of the cervical medulla. Further down, in the peripheral portion of the right lateral column, a transverse section revealed a spot of gelatinous appearance, about the size of one square mm. (one-twenty-fifth inch square), which could be traced down through the dorsal medulla to the lumbar enlargement, where, however, it was situated more towards the posterior part of the lateral column. The lower part of the cord was remarkably soft in several spots. The roots of the nerves showed no macroscopic alterations.

Cicatrices in the pharynx, liver and testicles. Spleen enlarged.

The *microscopic examination* of transverse and longitudinal sections of the hardened medulla, colored with carmine, revealed the following appearances:

A transverse section through the cord and the callous mass which was closely connected with it, at the point of adhesion, the upper boundary of which was just below the level of the lowest decussation of the anterior pyramids, showed, in the first place, that all the membranes of the cord were reduced to a coherent mass, in which the separation into pia, arachnoidea, and dura mater was no longer possible, and which completely filled up the subarachnoideal space. This mass consisted of ordinary fibrous connective tissue, which was traversed by greatly dilated capillaries and small arteries and veins, presented here and there accumulations of pigment arranged in serpentine figures, and, when the sections were tinged with hematoxylin, was found to contain great numbers of nuclei. In some places the vessels were surrounded by numerous round cells, and here the appearances of granulation-tissue were presented. This mass was intimately adhering to the medulla, over the entire surface of the posterior columns, as well as over the posterior part of the lateral columns, to a similar extent on both sides. The mass gradually decreased in thickness at the edges of the adhesion, the fibrillæ of the connective tissue grew more delicate, and the callosity was replaced by the normal pia mater over the lateral surface of the lateral columns. The line of demarcation between the callosity and the medulla was marked at the site of the adhesion by a darker contour and by large vessels running parallel with the surface. The posterior columns at this spot were transformed into a homogeneous tissue, which was colored evenly by carmine, but in which numerous nuclei were brought out by tinging with hæmatoxylin. This tissue extended from the periphery inwards, in thick, radiating lines, terminating at the posterior commissure, and laterally at the median substantia gelatinosa of the posterior cornu. Between these lines transverse sections of nerve fibres were seen, which were very few in number in the right posterior column, but were more numerous and were still united in bundles in the left. The posterior roots passed through the homogeneous mass. In the most posterior parts of both lateral cords there was a marked increase of the sustentacular connective tissue of the medulla, with a diminution in size of the transverse sections of the nerve fibrillæ. The greater part of the transverse sections of the lateral cords, the processus reticularis, and the lateral substantia gelatinosa presented, on the other hand, a normal appearance, and the spongy substance of the posterior cornua, also, with their cells, did not seem to be involved in the morbid process. The large cells of the anterior cornua, as well as the anterior pyramids, did not show any noteworthy alterations. The whole gray substance, however, as well as the degenerated posterior columns (particularly the right) was riddled with unusually numerous and large vascular openings, to such a degree that it actually presented a porous appearance. The transversely divided blood-vessels themselves had nearly all fallen out during the section of the cord; but, wherever these were preserved, they exhibited no remarkable dilatation, and there was no proliferation of nuclei in the neighborhood, the principal dilatation having taken place in the perivascular spaces. These alterations in the cord were found in all the transverse sections, which were made through the place where adhesion had occurred.

At the spot where the spinal marrow merges into the medulla oblongata, the degeneration was confined to the fasciculi graciles; the restiform bodies were normal; the pia on the posterior surface was thickened, but was not adherent to the dura mater. In the medulla itself the transverse sections of the columns and the groups of ganglion cells showed no apparent change; but even here, especially in the substantia reticularis, the same increase of the transversely divided vessels and dilatation of the perivascular spaces were found as in the gray substance of the spinal cord. The central canal in the cervical region was obliterated by an exuberant proliferation of cells, which extended as far as its termination at the floor of the fourth ventricle. Unfortunately, the rest of the spinal cord, in the process of hardening, was spoiled for microscopical examination.

In this case, the many interesting points of which cannot be discussed here as thoroughly as they deserve, the anatomical alterations manifestly present the residuum of syphilitic infiltration of the membranes of the medulla spinalis. The whole nature of the alterations in the spinal cord itself indicated that they were secondary to a morbid process developed on the surface; that there was an extension of the morbid process from without inwards, analogous to what occurs, as a rule, in like affections of the brain. The long duration of the disease and the often repeated treatment had undoubtedly led to a retrogression of the infiltration, resulting in the formation of cicatrices, which caused a marked and permanent interference with the functions of the nervous system, but left the patient still in possession of partial functional power, and probably would not have proved fatal if the intercurrent pulmonary affection had not carried off the patient.

According to our views, a case like this would belong properly in the first category, among the syphilitic new-formations, if it were not for the fact that cicatrization had already set in.

3. *Simple softening* of the cord is also mentioned, especially by Steenberg,[1] as a lesion found in some cases at the autopsies of syphilitic patients, who died of diseases of the nervous system. These, however, were cases in which there were either other complicating diseases, or in which the symptoms during life did not point exclusively to an affection of the spinal marrow, or, finally, in which the autopsy was not held till a comparatively long time after death. These reports, therefore, need revision. It is also asserted that genuine myelitis may be developed under the influence of syphilis; but we possess no conclusive instances of such an occurrence; the same may be said of the primary degeneration of the posterior cords. (As an instance of the *secondary* degeneration, see the above case.)

4. The *absence of anatomical lesions*, at least of such as are readily discernible, has also been repeatedly observed in syphilitic diseases of the medulla spinalis, *e.g.*, by Zambaco,[2] Kussmaul,[3] Leon Gros and Lancereaux,[4] etc.

The course of the affection in these cases resembled that of the acute ascending spinal paralysis, which has been described especially by French authors (Landry and others); it was very rapid, terminating in death within a few weeks. The autopsies furnished only negative results, although the examinations were most critical, and, in Kussmaul's case, was microscopic as well as macroscopic. We have here the analogue of an affection of the brain without anatomical lesions, which has been already discussed in detail, and the remarks made there are also applicable here.

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[1] l. c., Case 5, 33.

[2] l. c., Obs. 71, 72.

[3] Ueber den Mercurialismus, S. 369, ff.

[4] l. c., Obs. 86.

Symptoms.

The syphilitic affection of the cord begins usually in the later stages of the disease, several, and even many years after the first infection. Generally a series of external manifestations, the traces of which may frequently still be found on the bodies of the patients, and sometimes the signs of a far advanced cachexia, great pallor, extreme emaciation, and marked debility, have preceded the appearance of the nervous symptoms subjectively. The patients are wont to complain of a general languor and an indefinite feeling of sickness, which they cannot localize, for a long time before more definite symptoms occur. Then, simultaneously with the beginning of the disease in the membranes, symptoms of irritation set in, which may last for months without paralysis. Pains are complained of, sometimes situated at a fixed spot on the spinal column, in the cervical, lumbar or sacral region, where they may be increased by pressure; sometimes situated in the extremities, where they are usually localized, at first in one arm or one leg, but later on involve some other limb. These pains resemble neuralgic less than rheumatic pains, extend over several nerve trunks, and are subject to great variations in intensity as well as in duration. They are accompanied by *parœsthesiæ*, formication, tingling, and a numb sensation in the affected extremity, although the power of perceiving external sensible impressions is not necessarily disturbed. At the same time, motor disturbances appear, such as *rigidity*, *stiffness*, and even temporary contractions of certain groups of muscles or whole extremities. These symptoms are ushered in by stiffness of the neck, opisthotonus, and pain in the contracted muscles. The stiffness of an extremity causes a sensation of difficulty in locomotion and of debility. Painful cramps are excited by movement. In one case I observed that, for many months, a convulsive, and, as long as the limb was unsupported, continuous tremor of the leg, which was, however, already paralyzed, set in whenever passive movement was attempted. These symptoms also are subject to great fluctuations. They come, go, and return again, often under the influence of accidental causes. This stage, which may be called prodromal, or, more properly, meningitic, may continue for weeks, and may be interrupted by intervals of several months' duration, during which the health is apparently undisturbed.

Finally, however, a new exacerbation takes place, and paralyses are added to the symptoms of irritation. The patient first notices a rapidly increasing weakness in one leg, or (if the seat of the disease be in the cervical region of the cord) in one half of the body, and suddenly, some morning, finds himself unable to move the affected limbs. It is characteristic of this affection that, after the symptoms of paralysis have once made their appearance, they attain a high grade in a remarkably short time. Before long the other extremity or half of the body will be affected, though with somewhat less intensity, and the paraplegia will rapidly become complete. Its extent will depend upon the seat of the lesion. When this is located in the lumbar region of the cord, both lower extremities will be paralyzed, but one usually much more so than the other; when it is located in the lower part of the dorsal region of the cord, the sphincters will also be involved in the paralysis. After the disease has attained a certain height, it remains stationary for a considerable time, and the patient is confined to his bed for weeks and even months. It must be stated here that the disturbances of sensibility do not usually increase in a corresponding ratio with the motor paralysis, as they do in other tumors of the cord or in myelitis. The symptoms of irritation, the pains, formication, etc., continue for a long time during the stage of paralysis, but the anæsthesia is wont to be much less prominent; the sense of touch, the power of distinguishing heat, etc., are preserved unimpaired, as, *e. g.*, in the case detailed above, or are diminished only at a few circumscribed points. The stationary period may have different terminations. Under energetic and persevering treatment the cases may very slowly improve and terminate in comparative recovery. In this connection the most favorable cases are those in which the infiltration and meningitis are confined to the lowest part of the cord. A gradual improvement of the paralysis, particularly in the less affected extremity, will take place, and even the more affected one will regain to a certain degree its functional power. But there remains permanently a more or less marked debility, a diminished usefulness of the extremity, which is consequent on the unavoidable cicatrization within the cavity of the dura mater. The outlook is less favorable when the lesions are so situated that they affect the sphincters of the rectum and the bladder. In this case, after the stationary period has lasted for a variable time, disturbances in the nutrition of the integument of the nates and bed-sores supervene, which rapidly increase and lead to the well-known dangerous consequences, or a cystitis is developed. When one of these complications sets in, the course of the disease, which had been previously entirely free from fever, is marked by irregular febrile action of a remittent character, which may attain a high grade in the course of time; loss of appetite and other disturbances of digestion make their appearance, and a fatal termination is ushered in by general debility and exhaustion.

The patient's condition becomes critical in a much shorter time when the meningitic infiltration is localized high up on the spinal cord, and especially when it is situated in the highest part of the cervical region. In that case the paralyses of the muscles of the body in general, and particularly of the muscles of the trunk and of respiration, are developed in the course of a few days, with the same rapidity as the paralysis of the legs, described in the last paragraph. Under certain circumstances the clinical picture of an ascending paralysis may be presented: as the syphilitic infiltration on the periphery of the cord increases in amount, to the meningitic symptoms are added first a paraplegia of the lower extremities, next paralysis of the sphincters, then paralysis of the muscles of the trunk, and lastly paralysis of the arms and the diaphragm. Even in these cases, however, the course is usually less regular. The loss of power may be developed, first, as in the case detailed above, in an arm, which had been previously the seat of the principal pain; then the lower extremity of the same side, then both extremities of the other side, and at last the sphincters and the muscles of the abdomen may be affected. The evacuation of the urine is now interfered with, and at this stage already inflammation of the bladder and bed-sores in different situations may be developed. The expiratory powers are diminished, and the cough grows weaker, and becomes at last impossible; next paralysis of the intercostal muscles supervenes, and the thorax can no longer expand sufficiently in inspiration; this symptom may be more marked on one side of the thorax than on the other. And, lastly, if the points of origin or the roots of the phrenic nerves be involved in the infiltration, the action of the diaphragm becomes insufficient, and the patient may die of suffocation; or a rapidly developing pneumonia, or pulmonary gangrene, may terminate his life. The order of succession of these manifestations here evidently depends upon the order in which the bundles of motor fibres that lie close together in the highest part of the cervical region of the cord are compressed, or are rendered unfit to perform their functions by the inflammatory dilatation of the vessels, by softening or swelling, or are destroyed by the infiltration. This order must necessarily be different according as the infiltration begins upon the posterior or the anterior surface of the cord, and according to the varying depths to which the alterations of the neuroglia extend into the cord in individual situations. The characteristic features common to all cases, however, is—as has been already pointed out—the *rapidity* with which the individual paralyses follow one another, until the complete picture of a general paralysis, fraught with danger to life, is presented. A second characteristic feature is the presence of the symptoms of *meningitis*, which precede and also accompany the paralysis. Even when the disease has attained the height first described, it may be arrested by energetic treatment, although a complete cure is of course out of the question. In such an event, the infiltration cicatrizes, and the callosities left by it, and also the cicatrices in the cord which resemble gray degeneration of different parts of the white substance, constitute permanent organic lesions, to which ascending and descending secondary degenerations of the columns may in the course of time be added. When improvement sets in, it commences in the muscles of the trunk, and somewhat later the extremities regain the power of motion in an inverse order of succession to that in which they were affected; the patient is again able to walk, and he regains the use of his arms; but the recovery is incomplete. When the primary and secondary degenerations are confined to the posterior columns, the different manifestations of tabes dorsalis, and especially well-marked ataxia, may set in. The genesis of "syphilitic" tabes is, however, entirely different from that of the ordinary form, and the two diseases must not be considered identical.

All these affections of the cord, may—as happened, for instance, in the case observed by Engelstedt—be complicated by syphilitic neoplasms in the brain, or in the arteries or nerves of the brain; in that case the clinical picture of the disease will be obscured by paralyses of individual cerebral nerves, or by the occurrence of epileptic attacks, etc.

The clinical history and the course of the affection differ from those described above in those cases which belong to the last of the four classes mentioned in describing the anatomical lesions, namely, those in which the post-mortem examination has as yet failed to demonstrate any alteration either in the marrow or in the brain. We have here also to deal with a very rapidly developing malady—in fact, with *the most acute of all the syphilitic affections of the spinal marrow.*

This form, which resembles closely the acute ascending spinal paralysis, is distinguished at the outset from those already described by the fact that it makes its appearance at a *much earlier stage* of the syphilis—usually within the first year after the infection—during the existence of the earlier secondary symptoms, namely, the cutaneous eruptions. This affection is not ushered in by prodromal symptoms, and the meningitic symptoms, so common in the cases of the first categories, are also wanting. At most, in the cases hitherto recorded, on the day on which the paralysis appeared vague pains were complained of in different parts of the trunk or extremities. The paralysis is marked from the very beginning; it is generally a paraplegia of the lower extremities, but one arm and the opposite leg may be first attacked (as in Kussmaul's case). This paralysis is accompanied by a sensation of tingling and numbness, formication, etc., in the affected extremity. Noticeable disturbances of cutaneous sensibility are, however, wanting. Sometimes the paralysis of the muscles is preceded by a certain degree of weakness in the evacuation of urine, incontinence or anuria. When loss of power in one limb has once begun, it rapidly increases in severity, and also extends with equal rapidity from below upwards, or from one side to the other, and becomes complete in a very short time. Thus, in Zambaco's case, the patient, a man twenty-eight years of age, after a good night's rest, found, in the morning, that he was weak in the lower extremities ; at noon he was unable to void urine ; on the next day his urine was passed involuntarily, and on the following day he was unable to walk. It has been repeatedly observed, however, that, notwithstanding the intensity of the paralysis, there was no corresponding loss of sensibility. In one case only was there a question even of a diminution of the sensibility. *Not the slightest mention* has been made *of pain* in the spinal column, or of rigidity or contractions of the muscles. After the paralysis has thus in a few days been developed to the highest degree in an individual previously, to all appearances, healthy, it remains stationary for a short time, without—as it seems—involving the respiratory muscles. But now the danger approaches which threatens the patient's life ; already in the second week a bed-sore begins to form, which rapidly increases in extent and in depth, and leads to septic infection and death, which takes place in the third, fourth, or, at the latest, in the sixth or seventh week. This is the most exact clinical history that can be drawn from the few cases of the affection which have thus far been published. However, the characteristic differences of this form from those previously described may be perceived even from this necessarily fragmentary description. Recovery from this syphilitic spinal paralysis seems, according to our present experience, to be out of the question. Zambaco administered to one of his patients iodide of potassium in large doses, to the other liquor of van Swieten, ten grammes (not quite three fluid drachms) daily, without success.

Syphilis of the Peripheral Nerves.

Pathological Anatomy.

The nerves may be affected in various ways under the influence of syphilis.

In the first place, the affection of a nerve may be of a secondary nature, and possess no specific characters, in the cases in which the mere *pressure* of a syphilitic neoplasm leads to irritation, and, later on, destruction of the nerve-trunk. This is the case when a syphilitic exostosis is developed in a canal through which a nerve passes ; and the same may also happen sometimes when the ordinary syphilitic gumma grows around a nerve or compresses it against a bone, etc. In such cases, also, an ordinary neuritis may be developed with subsequent atrophy of the affected part of the nerve, just as it may be excited by pressure of other tumors. This occurs most frequently in the cerebral nerves during their intracranial course. It may be mentioned, here, that a nerve has sometimes been observed to pass through the centre of a neoplasm at the base of the brain, and nevertheless present no alteration. Generally the effects of the pressure are especially marked, when the tumor is developed on the dura mater, and a portion of a nerve—as, for instance, the ganglion Gasseri—lies between it and the bone. In such cases the affected nerve is found either slightly reddened and softened, and its sheath thickened (Courtin),[1] or it is *smaller* at the point of compression (Wagner),[2] or it is even atrophic (Virchow),[3] and transformed into a thin transparent band. No histological examination of such altered portions of a nerve has as yet been made ; but it is probable that the alterations in them are the same as are met with in the facial nerve in caries of the petrous portion of the temporal bone, viz., inflammatory infiltration of the connective-tissue constituents of the nerve, with disappearance of the tubular nerve fibres. In other peripheral nerves than those within the cranium this syphilitic neuritis from compression has been seldom observed. Zambaco reports only one instance of it.[1] In that case a syphilitic tumor as large as a walnut was found situated deeply in the left nates, underneath the muscles and compressing the sciatic nerve.

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[1] *Leon Gros* and *Lancereaux*, Obs. 133.

[2] l. c., Obs. XIII.

[3] Gesammelte Abhandlungen, S. 414

Moreover, the peripheral nerves may be affected in another way by a gumma situated in their neighborhood, namely, by a direct extension of the infiltration into them. This, however, seems *to be possible only* where the nerve is not yet surrounded by a dense sheath, *e. g.*, at the points of origin of the cerebral nerves from the brain, and especially at the chiasma and the adjoining parts of the optic nerves. This form then also exhibits an unfortunate preference for the cerebral nerves. The process that takes place here is the same as that which has been already described in treating of the syphilomas of the brain; the granulation tumor takes its starting-point from the pia mater, extends principally along the blood-vessels to the chiasma, and the tissue of the latter is swallowed up in the tumor. It is then impossible to draw a sharp line of demarcation between the nerve and the new-growth of the pia mater; the tissue of the former is replaced by the grayish-red, gray, or yellowish caseous material, of which the whole tumor consists. Several cases of this kind are described by Virchow,[2] Graefe,[3] the author,[4] and others. Such a process necessarily destroys the functions of the affected nerves.

Thirdly, a *primary syphilitic affection* of the peripheral nerves, in circumscribed spots, or spread over a considerable extent, may also occur. This affection also has thus far only been observed in the cerebral nerves. The cerebral nerves consequently are in every respect more predisposed to syphilitic affections than the spinal nerves. Whether the latter are altogether exempt from them is still questionable. The majority of observers only mention the occurrence of these primary degenerations of the nerves *within* the cranial cavity. The only exception to this is a case recorded by Essmarck and Jessen, in which the right oculomotorius was degenerated *externally* to the skull, while the left oculomotorius was similarly affected within the cranial cavity. These syphilitic degenerations of the nerves are found either along with gummy tumors of other parts of the contents of the cranium, or *without* such tumors, or at least their whole aspect shows that the disease of the nerves was the primary affection, and that the syphilis of the cerebral arteries, for instance, was secondary. The nerves have lost their naturally rounded shape, as well as their white color, over a greater or less extent. They are transformed into reddish connective tissue like or yellow cartilaginous masses (Dixon[1]); or into lardaceous cords, which are three times as thick as the healthy nerves (Essmarck and Jessen[2]); or they are swollen and infiltrated with reddish material (Virchow[3]); or soft, pulpy, and also reddish (Courtin[4]); or partly thickened and infiltrated with a grayish-yellow mass, partly already reduced in thickness, or entirely destroyed, so that the continuity is only preserved by the sheath of the nerves (the author[5]). In such cases the mere external appearances show that here, as in syphilis of the brain and the spinal cord, there is an infiltration of the nerve with a foreign mass—a neoplasm—which is here also sometimes reddish-gray, sometimes cheesy and yellowish, or lardaceous in appearance on transverse section. Sometimes both forms are found in the same nerve. In general, the infiltration adjusts itself to the shape of the nerve, the sheath of which is usually not perforated, and only presents knobby protuberances at various points. On the sections nothing of the original tissue can be seen. The histological investigation, made by Dixon in one of his cases, showed that the cartilaginous mass was composed of fibres and a granular substance. In the case published by the author, which has been already alluded to, the degenerated nerve, in which yellow masses were discernible scattered through a reddish-gray substance, was microscopically examined, and it was found that a very vascular substance, consisting of densely crowded spindle cells, had replaced the normal nerve tissue, of which only here and there a medullary fibre, surrounded by the neoplasm, remained. Probably a larger number of axis cylinders, deprived of their envelopes, were also present. The yellow masses were located partly in the substance of the neurilemma, partly on its inner surface, and consisted of a conglomeration of densely crowded granular cells, often of considerable size. It follows then that the nerve was replaced by a new-growth, which was anatomically perfectly analogous to the syphilitic granulation-tumor, and that therefore the term syphiloma, or gumma of the nerve, or, if it be preferred, syphilitic neuritis, is correct.

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[1] l. c., Obs. XXXIV.

[2] Natur der const. syphil. Affectionen. S. 83.

[3] Archiv f. Ophthal. VIII. 2 Abth. S. 74.

[4] l. c., Fall 45.

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[1] Medical Times and Gazette. 1858, Oct.

[2] Allgem. Zeitschrift f. Psych. 1857. S. 20.

[3] Natur der constit. syphil. Affectionen. S. 83.

[4] Arteriensyphilis. S. 70.

[5] *Leon Gros* and *Lancereaux*, Obs. 133.

Finally, it may occur that the peripheral nerves, or their nuclei in the central nervous substance, or their roots, or the ganglia, which are dispersed along their course, are altered during the existence of constitutional syphilis in such a manner that grave functional disturbances are excited, although it has been as yet impossible to demonstrate the alterations in question by means of the knife or the microscope. Thus, in this respect also, there is an analogy with syphilis of the brain or the medulla spinalis. Under this head belong perhaps the frequent neuralgias, and certain isolated paralyses of the nerves, which come and go in syphilitic individuals, without being preceded or followed by any other symptoms that indicate the occurrence of an organic degeneration of the brain or spinal marrow. Possibly the delicate alterations that occur in these cases may be discovered at some future time.

I may draw attention in this place to the communication of Petrow (l. c.), which, however, needs confirmation, on the more minute alterations in the structure of the sympathetic in constitutional lues. The author in question examined the sympathetic ganglia in a number of cases of constitutional syphilis, and found a more marked pigmentation and "colloid" degeneration of the protoplasm of the ganglion cells, and a proliferation of the cells that form the capsules, and also a transformation of the interstitial cellular tissue into a more rigid and dense mass, which compressed the nerve-cells.

Symptoms.

The isolated affection of one nerve is always revealed by a marked disturbance in its functions, while the neighboring nerves, or the contiguous portions of the brain-substance, are entirely unaffected. But when, in addition to the degeneration of the nerve, there is, as is more frequently the case, also an extensive gummy neoplasm at the base of the brain, the symptoms will be more complicated. We find, therefore, in the majority of the cases of affections of the cerebral nerves, in addition to circumscribed paralyses and pains, or anæsthesias, also the signs of the cerebral affection which has been already described. Sometimes, however, even after the development of an extensive anatomical lesion, the symptoms referable to a single nerve may remain for a long time perfectly isolated; and in fact there are cases in which the disturbances in the functions of a single nerve constitute absolutely the only symptoms of an intracranial lesion.

In such cases the functions of the affected trunk or branch of a nerve are by no means always completely arrested. Very often only a single muscle is paralyzed, while the other muscles, supplied by the same nerve, retain for a considerable length of time either their full or else a diminished power. There is an apparent incongruity between the anatomical lesions and the symptoms; for, while the paralysis has often been developed quite suddenly, or at least very rapidly, in the parts supplied by a nerve, the subsequent autopsy has proved that the affection of the nerve was not developed with the same rapidity. On the other hand, it must be remembered, that here, as well as elsewhere, the gummy neoplasm possesses the character of an interstitial lesion, which, for a certain length of time, leaves intact at least a portion of the normal tissues of the part in which it is developed. Thus an apparently completely degenerated nerve may still contain a number of conducting fibres, which may retain for a long time their irritability, but suddenly lose it rapidly and completely.

Of the cerebral nerves, the *oculomotorius* seems to be most frequently affected; and the first symptom is then usually *ptosis*, which occurs long before the alteration in the position of the bulb. This symptom is so common that the suspicion of syphilis is justifiable whenever a ptosis is developed without any other apparent cause, and in the absence of other symptoms. Later on, the paralysis of the recti muscles sets in, with consequent strabismus externus, prolapse of the bulb, and, finally, extreme dilatation of the pupil, with loss of the mobility of the iris. When the paralysis of one oculomotorius is entirely isolated, the face assumes a characteristic expression.

Next in frequency among the motor cerebral nerves, the *N. facialis* is affected. In this case the paralysis involves, in the usual manner, all the branches of the nerve; or only the branches which supply the parts around the eye are paralyzed at first. The clinical picture then presented is the same as has already been sufficiently described in this work, and a detailed description may be omitted here.

The *sixth cerebral nerve* is affected as frequently as the seventh, and oftentimes it is the only nerve affected. The symptoms are strabismus internus and diplopia, which appear either suddenly or gradually.

A circumscribed paralysis of the hypoglossus due to syphilis has not yet been observed. Unilateral paralysis of the muscles of mastication has, however, been observed in a case in which a general degeneration of the trigeminus involved also its motor root.

Hence the most prominent symptom of a syphilitic affection of a motor-cerebral nerve consists in the gradual or sudden occurrence of paralysis of one set of the facial muscles, which extends usually after a certain length of time to all the muscles supplied by the affected nerve. But if we consider that the anatomical process in the nerve—be this due to a compression of the whole nerve from without by a tumor, or to the growth of a neoplasm within its sheath—must always cause irritation and subsequent destruction of the nerve-fibres, it will be evident that the effects on the peripheral nerve-twigs and the muscles supplied by them must be the same as have been observed in any other neuritis, and have been described by Erb and others. Although these effects have not yet been demonstrated anatomically in these cases, the functional signs of this secondary degeneration of the nerve-twigs and the muscles have, nevertheless, been recognized in cases of syphilis. They are *loss of the electric irritability of the branches of the affected nerve, and atrophy of the muscles supplied by them.* Ziemssen,[1] in a case of this kind, has demonstrated the first symptom ; and the author himself, in a case of syphilitic paralysis of the facialis and ptosis, which had almost completely recovered, has had occasion to observe a considerable diminution in the contraction produced by the faradic current. The atrophy of the muscles is not usually observed, probably for the reason that those patients who do not recover perish by further complications before the atrophy has had time to develop. The attention, however, should be directed to this symptom, especially in cases which have imperfectly recovered. Lancereaux[2] mentions several cases of paralysis of individual muscles of the extremities, which, he believed, depended on a syphilitic affection of the nerves, and in which, after a long time, marked atrophy made its appearance.

Of the *sensitive* cerebral nerves, the *trigeminus* is most frequently affected, either during its course within the dura mater, or more commonly outside of it, in the Gasserian ganglion or in separate ramifications. Accordingly, sometimes the entire half of the forehead and face, sometimes only special parts of them, are affected. At first there are usually neuralgic pains, corresponding to the distribution of the branches of the nerve. These pains are subject to nocturnal exacerbations. Sometimes, as in the case of Graefe, the malady begins at once with anæsthesia. This latter symptom may be more or less complete, and is often accompanied by subjective pains in the parts deprived of sensation. Lachrymation and ophthalmia may easily be excited. When the lesion involves the whole of the fifth nerve, paralysis of the muscles of mastication sets in, preceding which spasmodic movements of the inferior maxilla have occasionally been observed (Lancereaux). With the exception of the nocturnal exacerbations of pain, there are no points of difference between this affection of the trigeminus and other non-syphilitic diseases of the nerve.

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[1] Virchow's Arch. XIII. S. 213.

[2] Traité de la Syphilis, p. 502.

The *affection of the optic nerve* is seldom isolated like those of the nerves already mentioned, but, under certain circumstances, it may also be the first symptom. When the affection is unilateral, the result is a rapidly developing amaurosis of one eye; when the chiasma is involved, there is an unsymmetrical dimness of vision in both eyes, with subsequent improvement in one eye. Hemiopia also may set in. The symptoms are aggravated by a consecutive hydrops of the sheath of the optic nerve. In these cases the so-called descending inflammation of the optic nerve can always be demonstrated by means of the ophthalmoscope.

A few cases of amblyopia and amaurosis, without demonstrable anatomical lesions, have been recorded, which were cured by anti-syphilitic treatment.

Besides the affections of the above-mentioned nerves, the literature of syphilis contains descriptions of a number of other nervous affections, especially neuralgias, which were held to be of a syphilitic nature, either because they were met with in syphilitic patients, or because they were cured by anti-syphilitic treatment. They do not, however, present any different symptoms from similar affections produced by other causes, and no anatomical proofs of their syphilitic origin have been adduced. It is possible that many of these cases are in some way due to the action of the syphilitic virus, but as yet there is no proof of it. As the symptoms of these functional nervous derangements do not present any characteristic peculiarities when they occur in syphilitic patients, I may content myself here with the simple enumeration of the most prominent of them. The one most frequently met with is sciatica; next in order come cervico-brachial neuralgia, occipital neuralgia, neuralgias of the testicles and of the scrotum, and, finally, visceral neuralgias, especially cardialgia.

Diagnosis.

The discussion of the diagnostic points, like that of the etiology, necessitates a general revision of the entire course of the disease; for it is of paramount importance that the existence of constitutional lues be demonstrated before an affection of the central or peripheral nerve substance can be pronounced syphilitic. It is true that the grouping, course, etc., of the symptoms referable to the brain and the spinal cord are sometimes so characteristic that the nature of the affection may be determined with a high degree of probability from them alone. It is also true that under certain circumstances the diagnosis of a syphilitic process may be made at the autopsy from the nature of the anatomical lesions found in those organs, even in the absence of other signs of syphilis; but such a diagnosis can only claim a more or less high degree of probability, and full certitude is only attained when the existence of the constitutional affection is proven by other symptoms. These symptoms are often very marked and characteristic, such as, for instance, more or less recent cutaneous eruptions, ulcers, or osseous defects in the nose, palate, etc., which place even the young practitioner upon the right track. The diagnosis, however, is not always so easily made. We have frequently had occasion to point out that the syphilitic affections of the nerves mostly belong to the so-called tertiary period of the constitutional disease, and hence it often happens that they only appear long after the superficial affections of the skin and the mucous membranes, and even the more deeply seated affections of the bones, etc., have disappeared. It is, therefore, necessary to search for the *traces of former* affections, for *cicatrices* and *defects*, when there is a suspicion that the affection of the nerves is of a syphilitic nature. The most important of these traces may be enumerated here, while the reader is referred to the comprehensive treatise on Syphilis in the third volume of this work for a detailed description of them. They are cicatrices of different forms on the genitals, cicatrices from buboes, pigmented spots of circular shape on the skin, cutaneous cicatrices of different sizes, especially white, depressed spots, as large as a lentil or larger, on the skin of the forehead or on the shin-bones, etc., where there are adhesions of the integuments to the subjacent bones; radiated cicatrices on the mucous membrane, especially of the mouth; rounded defects, that look as if they had been cut out of the arches of the palate, or the tonsils; irregularities of the surfaces of the bones, which present excavations surrounded by protuberances; a moderate but usually very hard swelling of the lymphatic glands, especially of the occipital, cervical, and cubital glands; enlargement and knobby induration usually of one testicle, or else atrophy of one testicle. All of these signs, or even a majority of them, are by no means always found in the same individual; it requires often a very accurate and close examination to find any of them. However, even a single characteristic cicatrix or enlarged gland may be of importance. If one such mark be found, the next object is to obtain by a detailed anamnestic examination an account of the course of the constitutional disease. The inexperienced practitioner, however, must always bear in mind that his questions will frequently be met by the most obstinate denials, and in such a case he will be obliged to content himself with the objective symptoms.

But even if the existence of constitutional syphilis, in an individual suffering from a nervous disease, be established, it is important to remember that it does not necessarily follow therefrom that the affection of the brain or spinal cord is also of a syphilitic nature. *A priori*, it is by no means impossible that a syphilitic individual may be afflicted with cerebral hemorrhage, meningitis, tabes dorsalis, etc., from the same causes which excite these diseases in non-syphilitic individuals. We should commit a grave error if we were to treat a melancholic patient, for instance, with mercury or iodine simply because he formerly suffered from syphilis.

It is then the task of the diagnostician to ascertain, from the nature of the symptoms, whether they could have been produced by a syphilitic affection of the nervous system. This also can, within certain limits, be decided. For, although every particular symptom of nervous affection may individually be caused by lues, the same does not hold true of every *complex* of symptoms. On the contrary, those which are due to syphilis of the nerves are marked by certain characteristic peculiarities.

If we consider, first, the syphilitic affections of the *brain*, we find indeed the most manifold forms of disease, but certain constant features are found in all of them. Common to all, in the first place, is the frequent fluctuation in the severity of the symptoms, which are at one time worse, and at another better. The improvement often takes place under a decided, specific treatment, but is occasionally spontaneous. It is sometimes so marked that the individual aggravations appear to be only so many relapses or episodes interrupting a condition of apparent health. The disturbances of the mind, the peculiar quasi-intoxicated state, the paralyses, the spasms, the pains, and the disturbances of the special senses may all be developed in this paroxysmal manner. The symptoms of other cerebral tumors, or of inflammation of the meninges, are not developed in this way. The progress of the former is always uniform and steady, and, although the outset of the latter may be sudden, its further course exhibits at the most only very transient amendments. A closer resemblance to cerebral lues is found in some cases of acute inflammation and abscess of the brain, which, however, very seldom take those repeated turns of improvement that are observed in syphilis.

Secondly, the grouping of the symptoms commonly bears a characteristic stamp. I may here refer the reader to the foregoing description, and will merely add a few examples, by way of illustration: *Psychical disturbances*, for instance, in a syphilitic patient, when really due to the syphilis, either assume the character of a dementia paralytica; or, if they appear in some other form, it is not uncomplicated. At the same time, uncommonly severe nocturnal headaches are complained of, or a unilateral loss of power, perhaps very slight in amount, is developed, or some of the cerebral nerves are involved, or, most frequent of all, epileptiform spasms are observed. Syphilitic *epilepsy* is always ushered in, or followed, by other symptoms. The intervals between the attacks are not entirely free from manifestations of disease; a complicating amaurosis or a ptosis is developed rapidly or slowly, or a sudden attack of hemiplegia supervenes. The *hemiplegic paralyses* of the syphilitic very frequently appear in the form of an apoplectic attack, so that we at first suspect hemorrhage or embolism. In such a case the age of the patient will assist the diagnosis. When a young person is suddenly seized with hemiplegia, or with aphasia and hemiplegia, and the examination of the heart and large blood-vessels does not reveal any abnormity, the possibility of a syphilitic affection must always be taken into consideration. Besides, this hemiplegia is frequently associated with a circumscribed paralysis of some cerebral nerve, and is followed usually by those disturbances of the intellect which we have described as quasi-inebriation, dreamy and typhoid, and which are observed neither in embolism nor in cerebral hemorrhage. Hence it follows that the way in which each individual symptom is *combined with the others* is often alone sufficient to indicate the correct line of investigation.

The characteristic marks of the syphilitic affections of the medulla spinalis seem, in the majority of the cases, to be the symptoms of spinal *irritation* with which they are ushered in, and the rapidity with which the paralysis increases; once it has made its appearance, even in cases where the symptoms of irritation have been wanting, the affection progresses with great rapidity in the form of an acute spinal paralysis.

Finally, with regard to the peripheral nerves, when these are the seat of neuralgic affections, the principal weight must be laid upon the nocturnal occurrence of the attacks; when they are the seat of paralyses, upon the restriction of the same within very circumscribed limits, and the rapid diminution of electric irritability of the parts involved; and, lastly, upon marked partiality of these paralyses for the nerves of the muscles of the eye (ptosis, strabismus).

Prognosis.

The syphilitic affections of the nervous system are among the gravest of the maladies which lues is capable of producing; and in a not inconsiderable number of the fatal cases of syphilis they are the immediate causes of death. The functions of the brain and spinal cord, moreover, are often irreparably impaired by the anatomical lesions produced by the syphilitic processes.

Nevertheless the prognosis cannot be considered absolutely unfavorable in these maladies; on the contrary, it is relatively good in certain cases, and is even more favorable than in most other organic diseases of the central nervous system. It cannot be doubted, although it has not yet been positively proven, that the syphilitic neoplasms within the cranium and spinal canal undergo a retrogressive process, atrophy, and cicatrization, just as the cutaneous tubercles and osseous gummata. Whether this is possible in syphilomata, that have undergone caseous transformation, is certainly questionable; but at least it is in the highest degree probable that it occurs in recent formations. Cases like the one described by me above, with the callosity in the membranes of the cord, or those which follow a similar course to what is observed in the affections of the arteries (change of the neoplasm into fibrous cellular tissue with simultaneous diminution of volume), render this theory very plausible. It is true that the new-growths in the membranes of the brain, etc., seem to resist specific treatment much more obstinately than do the external affections. Not unfrequently cases are observed in which the eruptions and cutaneous gummata undergo a retrograde process, even entirely disappear, and yet death supervenes soon afterwards, and the post-mortem investigation reveals neither arrest nor retrogression of the internal lesion. At any rate, a much longer time is required for the cure of the latter than for the cure of the soft gummata of the periosteum or other places. It must further be borne in mind, that even when the new-formation has cicatrized, there is not a perfect restitutio in integrum, not a complete disappearance of the growth, but that invariably a more or less extensive cicatrix remains, which may cause permanent functional disturbances. In the majority of those cases, therefore, in which the very grave symptoms disappear, we may be justified in giving a good prognosis so far as the preservation of life is concerned, but the health will never again be as perfect as before the attack. A certain noticeable deficit in the functional powers will always remain—always provided, that the symptoms were actually caused by neoplasms somewhere within the cranium. The prognosis will be most favorable, when the new-growths are situated only in the dura mater, or in circumscribed spots between the membranes, and do not directly involve the nervous substance.

Of those cases which run their course "seemingly without actual lesion" (sine materia), only a few, at least when the brain is involved, terminate favorably. Hildenbrand (l. c.) has observed several of these cases recover under proper treatment. The acute ascending spinal paralysis in syphilitic individuals, however, seems invariably to terminate unfavorably.

In a prognostic sense, the individual symptoms are of very varying importance, the value of each evidently depending upon the seat and extent of the lesion to which it usually owes its origin. The simple *psychical disturbances*, and also the *epilepsy*, if there exists no complication on the part of the cerebral nerves, etc., admit a comparatively favorable prognosis. In such cases we have perhaps to deal with very small exudations on circumscribed points of the periphery of the brain. Many of these cases are known to have recovered. The paralysis of a *cerebral nerve*, or the *circumscribed neuralgia* or *anæsthesia* of the same, is not in itself of very grave prognostic significance; for here too the part involved may be of very limited extent. We must, however, express ourselves very reservedly concerning the prospects of ultimate cure, when the paralysis is already established. For, even in case of recovery, the cicatrization of the infiltration is sure to leave defects in the affected nerve, which will interfere with the complete return of mobility. Only in the very earliest stages of the affection can a complete recovery reasonably be expected; and usually the patients do not come under the observation of the physician until the paralysis is well marked, and often not until it has existed for a long time. But when, in addition to a circumscribed paralysis of cerebral nerves, general cerebral symptoms, such as convulsions, sopor, or hemiplegia (usually of the opposite side) appear, the prognosis becomes at once very dubious; for now the complex of symptoms indicates that the new-growth has involved a considerable portion of the base of the brain, and especially that the arteries are in danger, or have already been involved in the morbid process. The degeneration of the cerebral arteries necessitates, under all circumstances, a very doubtful prognosis. The hemiplegias in particular, which are dependent on the disease of the arteries, are, with few exceptions, permanent and incurable, because we have to deal here with a destruction of important motor provinces of the brain. When there is no hemiplegia, but only the peculiar quasi-intoxicated condition, the prognosis is by no means absolutely bad, even if the symptoms be apparently very grave. For in this case, even after the somnolency has lasted for weeks, with delirium and confusion of mind, a marked improvement and even apparent recovery may take place. A certain defect in the intellectual functions will, however, always remain.

With regard to the *affections of the spinal cord*, the occurrence of pains in the vertebræ and in the extremities, of muscular rigidity, and of paræsthesiæ, should at once warn the practitioner of the necessity of caution in the prognosis and promptness in the treatment. The prognosis becomes doubtful as soon as paralyses appear; but even then a certain degree of cure is still attainable.

Treatment.

The discussion of the treatment of the above-described affections requires but few words. We consider it unnecessary to discuss here the question, so often debated in former days, whether a previous treatment of the syphilis with mercurials may possibly be the cause of these nervous symptoms. There are but few physicians nowadays who hold this view. It is only necessary, when any one is still in doubt on this point, to refer him to a comparison of the above description with the symptoms of mercurialism, as given in the already mentioned work of Kussmaul; or it will be sufficient to point out to him that at the post-mortem examinations of cases of mercurial poisoning no lesions bearing even the remotest resemblance to the new-growths in the central nervous system, described in detail above, have ever been found.

We know that the latter are identical with the external affections produced by syphilis, which can be cured by the administration of mercury and iodine. It is therefore evident that the syphilis of the nerves in all cases calls for the administration of these antisyphilitic remedies. The numerous cases of recovery reported have, with hardly a single exception, been treated in this way. In view, however, of the great danger which threatens the organism and life of a patient suffering from syphilis of the nerves, it is plain that the employment of these remedies must be begun as early as possible, and that they must be given *energetically, in large and frequently repeated doses*, for everything depends here upon speedily preventing any further extension of the malady.

This fundamental principle being settled, the physician has only to consider which of the two antisyphilitic remedies is to be employed in each individual case, and in what form it is to be administered. Lancereaux and Leon Gros are of the opinion that mercury is especially indicated in the secondary manifestations, the congestive and inflammatory affections, while iodide of potassium is the proper remedy for the tertiary affections, and particularly for the gummy infiltrations. It would be difficult, however, to carry out this separation of the symptoms in every case during life. Moreover, as we have already endeavored to demonstrate, we have probably, in the majority of the cases, to deal with the so-called tertiary processes, *i. e.*, infiltrations in the membranes, arteries, etc. It seems to us more suitable to consider that remedy the more valuable which the more rapidly and certainly arrests the gummy infiltration process, and causes it to take a retrograde course. When compared in this way, mercury seems in every case to deserve the preference. In all cases, therefore, in which the symptoms are urgent, such as intellectual disturbances of any kind, epilepsy, beginning paralysis, or, on the part of the spinal cord, pains in the vertebræ, and rigidity or commencing loss of power in the muscles, we always recommend first the use of *mercury*. Of the various methods of employing this remedy, that one is to be preferred by which, in the shortest possible time, the largest possible quantity of the drug can be incorporated with the body. Long experience has proved this to be the "inunction cure." This cure has the additional advantage of being much easier of application than the internal administration; it is also easily applied when the patient is unconscious or opposed to treatment. We must particularly insist on the necessity of disregarding, when called upon to treat a case of syphilis of the nerves, all those circumstances which usually counterindicate the use of the inunction cure; even if the patient be weak and exhausted, we must not hesitate to employ it.

The "indicatio morbi" here has the precedence of all other considerations. The danger increases daily, as long as energetic, radical treatment is delayed. Neither does the fact that the patient had, years before, or even only a short time previously, gone through an incomplete or complete course of the same treatment, furnish a counterindication to its repetition. Numerous observations, made in recent years, particularly in Aix-la-Chapelle, have demonstrated that mercury has lost none of its efficacy in cases in which previous syphilitic affections had been treated by it, but in which relapses had set in. The remedy, used a second time, proved equally efficacious. It is advisable to let the patient take warm or sulphur baths for a few days before the treatment is begun, in order to promote the capillary circulation of the skin; before each inunction also a bath should be taken, or the parts to be anointed at least washed off. The doses to be used in each inunction must be larger in these than in milder cases. We usually order, during the first fourteen days, from three to four scruples of mercurial ointment, to be rubbed in daily in the usual manner in the lower and upper extremities, the abdomen, and back successively. After this, if an amelioration of the symptoms be noticeable, the same dose is repeated every other day for several weeks, and then half this dose is used at the same intervals for several additional days or weeks, according to the course of the affection. With regard to the duration of the treatment, a special warning must be uttered here against a premature discontinuance of it; a return and an aggravation of the affection is too often observed shortly after an interruption of the treatment. In this connection Yvaren very truly says: "The treatment must be as obstinate as the disease." The physician must not lose patience. In general it is advisable to continue the inunctions at least fourteen days after the graver symptoms—such as the disturbances of the intellect, the loss of power, or the spasmodic attacks, etc.—have disappeared. Moreover, if there be the slightest indication of an aggravation of the affection, a new course of treatment must be commenced. The advice of Sigmund, to keep the mouth and teeth scrupulously clean, must be carefully complied with, in order to prevent salivation, which would necessitate an interruption of the treatment at too early a period. When the patients are unconscious, this duty will devolve on the nurse.

The diet to be employed during the continuance of the treatment will vary with the state of the constitution of the patient. When his strength is sufficient to bear it, we would recommend low diet, and even the "hunger cure" (a water soup and some biscuit three times daily) in connection with the inunction cure. We are less afraid of the syphilitic anæmia induced thereby, because we hope to avoid the main danger more promptly by obtaining more rapidly the effects of the treatment. Only very debilitated patients require a more generous diet during the treatment: meat broths once or twice daily, with a ration of roasted meat and some preserve at noon, and milk in the morning and afternoon.

If affections of the skin or other circumstances counterindicate the inunction of mercury, *injections* of corrosive sublimate may be resorted to in its place; but they must be employed persistently for a long time. There are cases on record which improved greatly under this treatment.[1] In one case, published by myself, this treatment was unsuccessful;[2] but it is possible that the patient was placed under methodical treatment too late.

Internal mercurial treatment has been frequently employed, especially by French physicians, in syphilis of the nervous system, and their success has been good. It may be recommended when the affection is of limited extent, and when the patient is only troubled with isolated symptoms, such as headache, insomnia, or epilepsy, and cannot be prevailed upon to submit to the inunction cure. Of the forms of mercury adapted to internal use, we may mention corrosive sublimate, calomel, and the green iodide given in the form of pills or powders. Rayer recommended particularly Sédillot's pills, which are composed of mercurial ointment, starch, and soap. In Lancereaux's work the administration of the liquor of van Swieten (corrosive sublimate dissolved in whiskey) is frequently mentioned in the records of cases. In general it seems that the quantities of mercury which can be taken internally without exciting marked disturbances of digestion, are insufficient, in the severer forms of syphilis of the nervous system, to cause the arrest of the affection in as short a time as is necessary.

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[1] *Heubner*, Arteriensyphilis, Fall 50. S. 119.

[2] Ibid. Fall 46. S. 69.

In such cases the iodide of potassium may be administered with more advantage. This remedy, which is slower in its action than mercury, is indicated where the affection is confined to the trunk or branch of one nerve; where a neuralgia, a circumscribed peripheral paralysis, or other isolated symptoms, point to a syphilitic affection of very limited extent; where the graver cerebral symptoms are wanting, and the disease in general exhibits the characteristics of a mild, slowly progressing malady; finally—according to Hildenbrand—in the cases which resemble general paralysis in their course. The small and irregular doses, however, in which it is not unfrequently administered, are entirely useless, and are even dangerous, because the patient is thereby encouraged in the idea that his malady is being cured, while it is in reality constantly but insidiously progressing. The drug should always be given in large doses, commencing with fifteen and a half grains a day, and increasing the amount by the same quantity every day, or every second day, until from one and a half to two and a half drachms are taken daily. It is most advantageously prescribed in a concentrated solution (eight scruples to four fluid ounces), of which from two to four tablespoonfuls or more are to be taken daily. For patients with irritable stomachs it is advisable to mix the tablespoonful of the potash solution with a glassful of Adelheid water.

In addition to the above-mentioned cases, the iodide of potassium may be used with advantage in those cases in which, in consequence of an energetic mercurial treatment, the most prominent symptoms of the nervous affection have disappeared; but certain effects of the disease, such as a paralysis, contractions, partial spasms, etc., still remain, of which it is uncertain whether they depend upon some residue of the syphilitic neoplasm or upon the cicatricial callosities resulting from it. In such cases we have continued the use of the iodide for months, frequently in conjunction with warm or steam baths, or sulphur baths, and have had the satisfaction of witnessing a constantly progressing improvement.

Besides these specific remedies, we must frequently resort to a *symptomatic* treatment for various troubles.

We are often called on to prescribe for *insomnia* in which chloral hydrate is of great service. When the *neuralgia* is very severe, it is often necessary to employ narcotics, particularly injections of morphine or atropine, as palliatives, while waiting for the specific remedies to exert their curative action.

In severe *congestions* of the head, a local abstraction of blood from behind the ear, or the persistent application of an ice-bladder, is sometimes of service. Never, however, even when the symptoms are of the gravest character, should we resort to general venesection ; for, in consequence of the great frequency with which the arteries of the base of the brain are affected, it is more likely that in these severe cases we have to deal with a deficiency of the intracranial circulation than with a plethoric condition. On the other hand, in the condition of protracted somnolence the repeated administration of stimulants, especially wine, and, under certain circumstances, even of medicinal stimulants, such as musk, is recommended.

In all cases care must be taken to keep the organs of respiration and digestion in good order. The bowels must be kept open by enemata or by aperient mineral waters. When the patient is in a state of sopor, the bed must be carefully attended to. As soon as the condition of the patient will admit of it, he should abandon the recumbent posture, and sit up occasionally for a few hours, and, when possible, walk around a little every day.

Finally, the *paralyses remaining* after the recovery are to be subjected to particular methods of treatment, which, if continued persistently for months, and even years, are frequently attended with some success. Of these we may mention daily gymnastic exercises of the affected limbs ; massage and frictions of the limbs ; rubbing with spirituous liquors ; finally, the persistent and methodical application of electricity. In hemiplegia and paraplegia both faradisation of the paralyzed muscles and the galvanic current, passed through the head and along the vertebral column, should be employed; in the peripheral paralyses of the cerebral nerves the application of the constant current is recommended. All these methods of treatment should be employed daily, or every other day, and be continued for a very long period. By these means atrophy of the muscles is prevented, and in the course of time perfect mobility of the affected limb is often re-established.[1]

Moreover, as a sort of "after-treatment," a methodical use of warm and sulphur baths, of baths containing carbonic acid, and even of sea-baths, combined with change of residence, is often useful. Such patients should be sent for several successive years to Teplitz, Wildbad, Aix-la-Chapelle, or Rehme; or to the East or the North Sea, according to the constitution of the individual. The cold-water treatment is also very properly recommended in these cases. The one unvarying rule for the employment of all these methods of after-treatment is, that they should never be begun until it is evident that the constitutional disease has been cured, and the anatomical lesions produced by it have cicatrized.

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[1] *Heubner*, Arteriensyphilis, Fall 47.

ACUTE AND CHRONIC
INFLAMMATIONS OF THE BRAIN
AND
ITS MEMBRANES.

HUGUENIN.

I. Inflammations of the Dura Mater.

The dura mater of the brain is composed of two lamellæ: the first, an outer or periosteal lamella, adheres more or less firmly to the bone (to which a number of vessels pass), and presents, on removal of the calvarium, a rough surface with numerous bloody points corresponding to the torn vessels. The adherence of this lamella to the bone is, in old persons, very firm—an explanation of which fact will be given later. In youth this membrane is not closely connected with the dura proper—a condition which often persists up to middle life, and for which no reason is known.

In this periosteal lamella the meningeal arteries ramify, and this membrane is therefore of the greatest importance for the nutrition of the cranial bones, and of the inner lamella of the dura, which contains but few vessels.

As the vessels of the second lamella (the dura proper) are few in number and of small size, it is whiter than the other, and tendinous in appearance. By folding on itself, it sends out prolongations into the cavity of the cranium from above and below, and from the two sides, viz., the falx and the tentorium, which are composed of this one membrane alone. The sinuses are formed by the separation of the inner and outer lamellæ. Both lamellæ are made up of dense, generally parallel, bundles of connective tissue, which, in many places, cross and interlace; it is not always possible, however, by the naked eye alone, to detect the spots where this interlacing takes place. There are also many elastic elements in both layers.

On the inner surface are several thicknesses of pavement epithelium. The existence of a layer which would correspond to the parietal layer of the arachnoid membrane of older writers, is denied by all later observers. The dura is supplied with blood by the meningeal arteries. These run in the periosteal lamella, send their branches into the bone on one side, and into the inner lamella on the other ; the latter branches are quite small.

The veins of the dura conduct the blood from parts of the cranial bones, and empty into the sinuses of the dura mater. These veins lie between the outer and inner lamellæ. This system of veins communicates with the superficial veins by means of certain branches (emissaria condyloidea and mastoidea, emissaria parietalia, the communicating branches which pass through the foramen cæcum, the canalis caroticus, the foramen ovale, the foramen hypoglossi, and those which communicate with the veins of the orbit).

The dura is supplied with nerves, some of which end in itself and its vessels, while others simply pass through it into the bone. These nerves, for the most part, follow the course of the meningeal arteries, and are as follows :

1. Nervus spinosus (Arnold, Luschka), from the third branch of the trigeminus ; it follows the course of the middle meningeal artery.
2. A twig from the second branch of the trigeminus (Arnold) ; it also follows the middle meningeal artery.
3. Nervus tentorii (Arnold) ; it is derived from the first branch of the trigeminus, and traverses the tentorium.
4. A nervus recurrens, from the vagus (Arnold) ; it supplies the region of the sinus transversus.
5. A branch of the hypoglossus ; it is given off in the canal of the hypoglossus, and pursues its course alongside of the posterior meningeal artery.

Sensitive filaments are not to be found in the dura ; yet nevertheless a decided sensitiveness to pain exists ; but, as is the case with other internal organs, an accurate localization of the pain is impossible. Pain is referred to quite a large area, even though the irritation be confined to a relatively small spot.

A.—Pachymeningitis Externa.

The causes of the inflammation of the outer lamella of the dura belong mainly to the department of surgery. Inflammation results from various causes.

1. From *injuries which cause the separation of the membrane from the inner surface of the skull.* The frontal, parietal, and temporal bones are the most frequent seats of this injury. As the amount of the consequent extravasation varies very much according to the size of the vessel ruptured, there results, in some cases, a tense tumor reaching inwards; in others, only a layer of blood between the dura and the bone. From the crowding of the cranial contents severe general symptoms may occur, and from the limitation of the pressure to one spot of a single hemisphere, symptoms characteristic of localized disease may follow. If the symptoms of general compression are accompanied by those of a local lesion, a diagnosis can be made. If, however, the layer of blood is thin, a diagnosis is impossible, from the insignificance of the symptoms.

This inflammation exceptionally increases to suppuration, which may involve a large surface, being attended with extensive separation of the dura from the bone. In this case a necrosis of the bone invariably follows the stripping off of the periosteum.

2. *From injuries, which cause perforation of the cranium,* either laying bare the dura, or, from direct action on the dura, causing contusions, or solutions of its continuity. Incised, punctured, and bullet wounds, as well as splinters of bone, whether attended or not by fracture through the whole thickness of the bone, may cause injury of the dura. This varies greatly with the nature of the case, sometimes there being a clean incision or puncture without bruising, and sometimes a tearing of the dura with severe contusion and consequent necrosis, if life continues for any length of time. The latter is most apt to occur in fractures of the base or of the convexity, with opening of the sutures.

3. *From the transmission of inflammation from neighboring tissues.* In these cases caries of the petrous portion of the temporal bone plays a prominent part, giving rise to inflammation of the outer lamella of the dura, and unfortunately not, as a rule, to that alone. The inflammation is always suppurative (except in a few cases where a chronic inflammatory thickening of the membrane takes place) before a perforation of the bone occurs.

Purulent inflammation of the dura follows not only carious destruction of the petrous portion (that is, purulent inflammation of the tympanic cavity and the labyrinth, with its sequelæ), but also carious destruction of the wall of the meatus auditorius externus. This is easily understood, if we consider the close proximity of the outer wall of the meatus aud. ext. to the dura, and that only a microscopic breach in the thin wall of bone, which forms the upper covering of the tympanic cavity, is needed to bring the pus from the tympanum in connection with the dura. The following are the ways by which suppuration usually extends to the dura:

a. From the upper wall of the external auditory canal (Toynbee); Troeltsch has also observed this.

b. Through the perforations in the roof of the tympanic cavity (a frequent occurrence).

c. Through the meatus audit. internus, which is lined by a prolongation of the membranes.

d. Through a perforation which may become established in the anterior wall of the mastoid cells.

e. Through a canal, which commences in the cells of the mastoid process, passes under the upper semi-circular canal, is continued on to the posterior surface of the pyramid, and opens into the sulcus petrosus superior, between the entrance of the aquæductus vestibuli and the porus acusticus internus on the corner of the pyramid.

f. Finally, Maas describes small bony canals through which, as he had observed in one case, inflammation may pass from the middle ear into the cranium. These are situated behind the superior semi-circular canal, and connect the middle ear with the middle cranial fossa. They contain, as he states, vascular prolongations of the dura; they are, however, inconstant.

In a similar way purulent inflammation of the outer lamella of the dura follows carious destruction of other cranial bones, viz., the ethmoid and the flat bones of the skull. Here specific disease of these is the most frequent cause. As a general rule, the inflammatory process does not for a long time go beyond chronic fibrous induration of the outer lamella, until suddenly, sometimes from no apparent reason, it becomes suppurative.

It must be remembered that caries or necrosis from any other cause can give rise to similar changes.

The question, whether inflammation on the outer surface of the cranial bones (inflammation of the mucous membrane in the labyrinth of the ethmoid, in the frontal sinuses, purulent inflammation of the galea) will not suffice to start in a short time, without caries or necrosis of the bone, inflammation of the outer lamella of the dura, must be answered affirmatively, according to our observations upon the purulent inflammation of the galea in erysipelas. The spread of suppuration occurs through the natural openings in the bones where vessels and bridges of connective tissue connect the galea with the outer sheath of the dura.

4. The *chronic fibrous thickenings* of the outer lamella of the dura, so common in old age, should be mentioned here. In advanced age the dura is always thickened, frequently dry and leathery, or whitish and tendinous. The arteries are found much thickened in their adventitia. In other cases the dura is found to have become firmly adherent to the bone, and this too without its having undergone any marked thickening.

5. The alterations of the dura which accompany *puerperal exostosis* (osteophyte) are also to be classed with the affections here under consideration. Their pathogenesis is not understood.

The anatomical appearances are, as would be inferred from the above remarks, very varied. It is certain, however, that inflammation, transmitted from without to the dura, almost never attacks the whole outer lamella, but is confined to circumscribed spots. These are usually smaller than the original inflamed area. If the inflammation is purulent, a purulent inflammation is usually transmitted to the inner lamella, though this does not necessarily take place.

The alterations of the dura are the usual ones: at first reddening in lines of various widths, corresponding to the congested blood-vessels, together with small punctiform extravasations, sometimes in great numbers; further, swelling and loss of consistency of the tissue, which becomes infiltrated with numberless white blood-cells collected in clusters and lines; and, finally, transformation of these into a tissue of spindle-cells seated on and within the dura, later developing into bundles of connective tissue. This leads to the chronic thickenings and deposits before mentioned. These are often followed by ossification—further details concerning which would be out of place here.

The macroscopic examination in some cases shows an adhesion of the dura with the inner surface of the cranium, with marked thickening of the former; in others, calcareous deposits in the young connective tissue, sometimes forming scales which are easily separable from the bone, and sometimes jagged points of bone-like consistency. The formation of true bone is not uncommon, causing a diffused thickening of the vitreous table, or the formation of rough bony projections, smaller or larger, as the case may be. This frequently happens in those traumatic extravasations with separation of the dura, when, on the inner side of the cranium, distinct traces of a wall-like ring of ossification are found.

If, on the contrary, the inflammation progresses to actual suppuration, other appearances are found, due to the fact that the wandering white globules, which at first were scattered in groups throughout the tissue of the dura, have been forced out upon its free surface, and have there formed distinct layers of pus. An active transudation from the vessels favors this, and white cells constantly leave the vessels, and active cell division takes place. The comparative lack of blood-vessels in the outer lamella may account for the fact that large collections of pus are seldom found.

Suppuration will take place if the dura becomes infected from a neighboring tissue. In such cases the tissue of the dura itself suffers important changes from suppuration; it becomes disintegrated, friable, soft, and often thinned to a web-like membrane. If the dura is examined in a later stage, it may be found that the abscess has been cut off from the inner layer by an active development of connective tissue. Much more frequently, however, the inflammation pierces through the entire thickness of the dura, and a pachymeningitis purulenta interna is added to the pachymeningitis externa. But this does not necessarily take place; sometimes the process stops with simple adhesion of the dura to the arachnoid and pia, or even to the surface of the brain. Even the last step in the inflammatory process, the growth of granulation tissue, may also take place under certain circumstances; it occurs when there is a defect in the bone (caries, fracture, with gaping or loss of substance), allowing the entrance of air. The hole can be closed by a process of cicatrization in the contiguous dura mater.

It is evident that the course of the inflammatory process will differ widely in different cases. Some cases end in death, preceded by severe symptoms; others run a chronic, non-suppurative course, and these during life are not characterized by any marked symptoms.

Mixed forms often occur, owing to the ease with which the suppurative process of the dura passes to the arachnoid and pia, and the close connection between this affection and inflammatory processes on the inner side of the dura. It is, therefore, often impossible to determine what is to be considered due to the inflammation of the outer surface of the dura, and what to inflammation in the parts beyond. The cases are very rare indeed where the disease can be recognized as a separate affection.

1. *Pachymeningitis of old age* is often discovered after death, without there having been a suspicion of the affection during life. In other cases the meningitis may have been accompanied by persistent dull headache; and at death, in connection with it, there may be found atrophy of the brain in every stage, with compensatory hydrocephalus, cloudy swelling, and serous infiltration of the pia. At the same time we have observed an equal number of cases where chronic headache was coincident with the above-mentioned alterations of the brain and the pia, without important alteration of the dura. It appears, therefore, fair to conclude that the common opinion, that the headache of old people is due to pachymeningitis, is not invariably correct; and, so far as we know, no distinguishing peculiarity marks those cases where a causal connection exists.

2. It is equally impossible to recognize in life the *puerperal exostoses* (Frankenhaüser); there are no symptoms on which even a probable diagnosis can be based.

3. *Purulent inflammation of the dura on its outer surface*,

following a wound, which had caused a hidden fissure of the skull, is by no means infrequent.

1. An epileptic patient, forty-two years of age, epileptic for seventeen years, fell and struck on a stone floor, causing on the left frontal bone a large contusion, which ran the ordinary course; but the patient did not recover entirely from the coma, and developed a slight hemiparesis of the right side, with paresis of the hypoglossus and oral and nasal branches of the facialis on the same side. There was also constant severe headache, especially on the side of the injury; but no fissure or depression was to be found; from the beginning, however, the sensorium was affected; at times the patient was not quite clear in his own mind as to where he was and what was going on round about him; decided loss of consciousness also occurred, and the patient vomited several times.

Duration of entire illness, eight days. During this time it was ascertained that the patient was laboring under a fever of very irregular type; in the morning the temperature was comparatively low; in the middle of the day it usually reached its highest point (sometimes as high as 103.5° Fahr. (39.7° Cent.)); while towards evening it fell again to 101.8° Fahr. (38.8° Cent.).

Slight clonic convulsions in the paretic limbs of the right side during the first four days, after which they ceased, only to give place to a somewhat more decided paresis of the extremities and of the regions supplied by the facialis and hypoglossus. During the first three or four days the pulse manifested the characteristics noticed in cases of pressure upon the cerebrum (from sixty to sixty-eight); during the last four days, after the convulsive manifestations had ceased, the pulse was less full and very frequent. The sopor became more and more marked, and coma finally ushered in death, which was somewhat hastened by a high degree of emphysema with bronchitis.

Autopsy.—A fissure extends from the middle of the left orbital region upwards to the parietal bone, and a short distance into it, traversing in its course the coronal suture.

Beneath the galea capitis, over and along the fissure, a thin layer of pus is visible. On the inner surface of the skull-cap the dura is separated from the bone over an oval-shaped space measuring one and a half by three inches. The sac thus formed is about three-quarters of an inch in width (depth, as it lies), and is filled with a chocolate-colored pus containing still a few blood-clots. That portion of the dura which forms one of the walls of this sac is thickened, soft, and in an active pyogenic state. On its inner surface the dura is infiltrated with pus at three different places, each of about the size of a lentil; and at these spots loose adhesions have formed with the arachnoid. A circumscribed portion of the outer surface of the arachnoid, not larger than a silver dollar, is covered with an extremely thin layer of pus; but in the meshes of the pia no pus can be detected—at least with the naked eye. Surface of the brain depressed throughout the region corresponding to the anterior central, and first and second frontal convolutions; brain substance dry and condensed, but manifesting no evidences of inflammation.

The case came under examination at exactly the time when the purulent inflammation was on the point of attacking the pia. The symptoms are not to be ascribed to the commencing leptomeningitis, but to the affection of the dura, in connection with the pressure on the frontal portion (motor centres) of the left hemisphere. This latter factor is by far the most important.

The following case shows that the symptoms are precisely the same in case of simple extravasation of blood without suppuration.

2. Epileptic, thirty-eight years old; an attack every three weeks. In the last the patient fell backwards, striking on the occiput; unconscious after the regular attack; sick twenty-three days, during which time the following symptoms were noted: From the very beginning total left-sided hemiplegia; left-sided ptosis; left-sided paralysis of the facial; and a slight paralysis in the rectus internus, causing a slight diverging strabismus. Finally, near the end of life, a paralytic dilatation of the right pupil occurred (oculomotorius). Sensibility on the paralyzed side not much impaired, but exact particulars not obtainable, owing to the patient's mental condition; no convulsions; moderate irregular fever, higher towards the end than at the beginning of the attack.

In the beginning pulsus cephalicus, small and frequent even on the fifth and sixth days. Nothing further of note until death; no convulsions. *Autopsy.*—Fissure of the cranium beginning at the right ear, traversing the right lambdoid suture, and extending through the occipital bone as far as the base. Very large extravasation of blood between bone and dura on the right side, nearly an inch in thickness, reaching forward from the extremity of the occipital lobe as far as to a point some little distance beyond the anterior central convolution, inward as far as to the falx, and outward to the operculum. Gray matter of brain in the condition of complete compression; also narrowing of the right posterior cornu; no suppuration, but superficial destruction of substance, particularly marked in the neighborhood of the fissure of Rolando.

If we compare the two cases, it will be seen that they differ only in the existence of convulsions in the first (the one in which there was suppuration). It is not possible, however, to ascribe the convulsions to suppuration. The absence of convulsions in the second case can be attributed with more probability to the total compression of the cerebral substance, which was not in a condition to respond to irritation. In case I.—*i, e.*, in a case of pachymeningitis ext. purulenta—there was no other symptom which could have helped to the diagnosis of the condition of the dura. We must, therefore, unreservedly agree with Hasse in the opinion that the severe symptoms, in cases of this kind, are due not to the affection of the dura, but to the simultaneous injury of the neighboring important organs.

4. The purulent inflammation of the dura, resulting from its exposure to the air by traumatic or necrotic destruction of a part of the cranium, has a very unimportant clinical significance. It is, however, a different matter if a carious process of the bone causes suppuration of the outer surface of the dura, and the formation of an abscess, which is liable to exert pressure, and which may at any moment cause the inflammation to spread to the pia. To recognize early such a serious condition, particularly on the petrous portion of the temporal bone, or on the frontal bone, would often be of the greatest importance. This, however, is not possible, especially on the petrous bone, where the abscess rarely attains any great size.

The sudden stoppage of discharge in otorrhœa is not a reliable symptom ; no one would be willing to conclude, from this, that the pus was discharging inwardly. It, however, should attract attention to a change in the condition of the affection.

A chronic otorrhœa can also give rise to purulent inflammation of the outer surface of the dura in one of the ways referred to above.

This can happen by perforation of the anterior and superior wall of the mastoid cells, without giving rise to a single external recognizable symptom of disease (either swelling or tenderness), as we have seen clearly demonstrated.

The headache, which often appears periodically in the course of caries of the mastoid bone, is very unreliable, and cannot be considered a symptom of a purulent inflammation of the dura.

This is also true of occasional attacks of dizziness.

The existence of this affection can therefore only be suspected. As soon as the external symptoms, however, are manifest, we may assume that both the outer and the inner surfaces of the dura, as well as the arachnoid and pia, are involved.

It is evident, from the above, that the physician could hardly be called upon to decide as to the prognosis of pachymeningitis externa by itself, since the underlying affections, and their most remote consequences, are comparatively of such overshadowing importance that it is to them that he would naturally look for the points on which to determine the merits of the case.

In traumatic suppuration the prognosis turns on the possibility of giving the pus, or the pus and extravasated blood, a sufficient escape. If opportunity is given for free discharge of pus, the affection of the dura often occasions but little trouble, as surgical experience has abundantly shown; it may, indeed, furnish material help towards cure by granulation.

If there is no opportunity for free discharge of pus, surgical experience teaches, with equal clearness, how severe an affection this is likely to prove, in the first place, by increasing the intracranial pressure, and, in the next place, by reason of the great rapidity with which it is apt to spread. The *prognosis* of the purulent inflammation of the dura in caries of the petrous bone is very unfavorable, as is shown both by post-mortem appearances and by clinical observation. Thickening of the pus may occur below the dura, thus affording at least a temporary check to the extension of suppuration to the pia mater. But it will be seen later, in the description of meningitis tuberculosa, how little room for hope there is if a cheesy mass remains beneath the dura.

Treatment.

As an accurate diagnosis of the affection is only exceptionally possible, but little can be said of any special treatment, which must follow the general indications presented by the underlying disease and its rapid extension. The treatment of meningitis, spreading from disease of the petrous portion of the temporal bone, will be considered hereafter. The question of trephining will arise in connection with the treatment of an extravasation of blood between the dura and the bone. This, however, belongs properly to the department of surgery, and will not be here discussed.

B.—Pachymeningitis Purulenta Interna.

We have seen that purulent inflammations, attacking the outer lamella of the dura, though interesting anatomically, are not of much importance clinically.

The same may be said of purulent inflammation of the inner lamella of the dura; it may lead directly to the incurable changes of the pia, so often mentioned above, but there is no purulent inflammation of the inner layer of the dura recognizable as an independent affection.

C.—Pachymeningitis Interna Hæmorrhagica. Hæmatoma Duræ Matris.

We will next examine the affection known as pachymeningitis hæmorrhagica, or hæmatoma of the dura.

From having been always regarded as accidental, and clinically but little understood, this affection has lately come to be classed among diseases which are capable of being diagnosticated. There is much discussion as to the origin of pachymeningitis, so that a somewhat thorough consideration of the question seems called for.

Pathological Anatomy.

This form of pachymeningitis presents itself as an organized mass situated between the dura and the surface of the arachnoid, and having different appearances in different cases. It may of itself, without any complication, lead quickly to death; while in some cases it is merely coincident with other fatal affections, not necessarily affections of the brain; finally, it may become chronic, and itself undergo many changes of character. Its localization even has long been a matter of dispute, partly on account of the changing opinions concerning the arachnoid and its relations to the dura and pia. The older writers (Abercrombie, Andral, Blondin, Rostan) held that the layer of blood (in the early stage of the affection the most noticeable feature) lay between the dura and the parietal layer of the arachnoid, the existence of which was at that time universally accepted. Houssard was the first (1817) to show that it was situated in the cavity of the arachnoid —that is, between the inner surface of the dura and the outer surface of the arachnoid (visceral sheath). Houssard believed that the arachnoid had nothing to do with the new-formation.

Baillarger (1839) came very near the truth in his description. In regard to the localization, he agreed with Houssard, and described the hæmatoma as a double membrane, in the cavity of which an extravasation of blood had occurred; he held, furthermore, that this blood was prevented from escaping by the adherence of the membranes to each other at their peripheries. He believed in the integrity of the parietal arachnoid. All later authors, with few exceptions, coincided with this view without important modification.

It was more difficult to agree on the origin of these membranes. Houssard believed that they consisted substantially of the fibrine of the extravasated blood; Baillarger and Boudet hold this opinion, as does Aubanel also, who, in spite of many errors, expresses very clear views upon the subject of the formation of these membranes.

Bayle insists, on the contrary, that from the very beginning the hæmatoma is an inflammatory product on the inner surface of the dura; while, in 1843, the French Academy came to no conclusion on the subject.

The Royal Medical and Chirurgical Society, 1845, did the same on the occasion of the reading of a paper by Prescott Hewett on the vexed question.

Engel (1842) and Rokitansky (1844) gave very good pathological descriptions, but did not attempt to explain the origin of the affection; while Durand-Fardel (1854), in a good treatise, speaks in favor of the belief of the original formation of a flat blood clot. In Germany, in 1855, more settled anatomical descriptions appeared. Heschl opposed the idea that the hemorrhage was the primary evil; he regarded the formation of the membrane as the primary manifestation of the affection, and this he describes as a connective-tissue growth rich in blood-vessels. He further called attention to the fact that the pachymeningitis, in the majority of cases, is situated on the upper part of the brain; while if an extravasation were the origin of the affection, the blood would sink to the base.

Virchow's well-known investigations (1856) were memorable above all others. His ingenious explanation was, that the affection is a hemorrhagic inflammation of the dura. The first product is formed from the dura, but all subsequent changes and deposits were developed from the new membrane itself. It is not possible to oppose other theories to Virchow's clear explanation excepting in regard to the genesis of the first changes.

A similar explanation can be found in Cruveilhier (Phlegmasie Pseudo-membraneuse hémorrhagique de l'Arachnoide parietale).

It is natural that most authors afterwards followed the views of the greatest of the promoters of modern medicine. Schuberg (1859) agrees entirely with Virchow; also Charcot and Vulpian (1860), Geist (1860), Guido Weber (1860). Griesinger does not touch the question; while Wagner (1868) and Kremiansky (1868) particularly endeavored, by experiments and new investigations, to give further support to the accepted theory. A few have advanced other views (Ramer, 1862; Sperling, 1872), but in general the explanation given by Heschl and Virchow has remained the accepted one up to the present time.

According to this the pachymeningitis begins with a hyperæmia of the dura—in the majority of cases, as Kremiansky believes, in the tract of the arteria meningea media (regio bregmatica); the arteries are enlarged, the hyperæmia of the capillaries is shown by a rosy flush of the inner surface. Then a loose, veil-like yellow coating appears, dotted with a number of confluent or scattered bloody points. It can be stripped from the tissue beneath, tearing many small vessels that enter the pseudomembrane from the dura. The membrane is quite rich in large vessels (hardly capillaries, Rindfleisch), usually three or four times as large as capillaries, with varicosities. The basis substance between the vessels, Rindfleisch describes as mucous tissue, composed of star-shaped connective-tissue cells. The subepithelial layer of the dura is the original source of the new-formation; wandering cells pass from the blood-vessels, and develop into a loose connective-tissue parenchyma. This delicate primary coating, often hardly thicker than a spider's web, is traversed by a number of vessels, and supplied with blood from the dura. From these vessels intercurrent hemorrhages arise, which are found in different quantity at different times, and belong to the anatomical appearances of pachymeningitis. These cause new-growth of membrane—which again is the source of new hemorrhage. So, finally, enormous masses of blood can be poured out between the thickened membranes, thus giving rise to numerous transitional forms between the pachymeningitis and the hæmatoma.

No modifications will be offered of the theory put forth to explain the later alterations of pachymeningitis; an attempt will, however, here be made to show that another explanation of the origin of the hæmatoma is possible.

We give first a description of the anatomical appearances from personal observation.

1. The *earliest stage* which presents itself to observation is as follows:

On the inner surface of the dura a thin layer of coagulated blood is found, of dark red color, thickest at the upper part of the cranium (2 mm.), and decreasing in thickness as it spreads in all directions, disappearing gradually at the edges without sharply marked border. This layer of blood is not entirely separable from the dura, but only in small membranous shreds, the consistence of which is very slight. The dura appears under this to be absolutely intact; there is no increased injection of the blood-vessels, no thickening; in places, which are handled with great care, the epithelium of the inner surface is clearly demonstrable. There are no connecting vessels between the coagulum and the dura; the coagulum itself shows no firm membranous portion. If the thinnest possible layer, taken from the surface of the coagulum, be placed under the microscope, only that is seen which can be found in any very thin layer of blood. Significant of coagulation, a delicate lace-work can be seen, woven with threads, forming a mesh-work containing masses of blood globules of normal shape, in places concealing the network. The condition of the white globules is of the greatest interest. By virtue of their disposition to unite with similar masses of protoplasm, they are found in groups sometimes making plates and rows, the latter of the width of three or four white globules, resembling the appearance of the laminated thrombus. But no organized element is to be seen, no connective-tissue cell, no vessel, nothing but the remains of coagulated blood. Further, neither the epithelium of the inner surface of the dura, nor that of the outer surface of the arachnoid, is in the slightest degree changed by the whole occurrence. The opportunity of observing this first stage of pachymeningitis occurs comparatively rarely, and then only in patients with dementia paralytica.

2. A *second stage*, of which many well-marked examples have been offered to us, presents macroscopically a different appearance. The formation on the dura resembles a membrane much more decidedly ; it still cannot be stripped in toto from the dura ; the color is brown or yellow, in some places reddish-brown, presenting an irregular, spotted appearance. In this stage also it is easy to satisfy oneself of the integrity of both epithelial surfaces. In no single case was it possible to find a marked injection of the dura in the tract of the art. mening. med., or anything resembling a true inflammation. The thinnest portions of the deposit present the following appearances under the microscope :

The main structure of the substance still consists of red blood globules and fibrine. It is easy to see that the blood globules gradually give up their coloring matter—at least this process may be seen to have begun in many spots ; in other spots the globules remain in the condition of relative integrity. But where the process has made further progress, the red globules lose their clear contour as well as their normal color ; masses of brown pigment appear in great quantities and irregular arrangement, lying between the masses of blood globules, which now resemble in their arrangement a fine network. The fibrine, which before was quite visible, disappears more and more from view ; what becomes of it cannot be said with certainty, but the colorless clumps of protoplasm, which were once red globules, are still distinctly visible. The white globules, however, whose peculiar arrangement was mentioned above, have suffered a characteristic change. They have begun to send out arms, in general from opposite poles, so that from the rod-like arrangement just mentioned bundles of spindle cells have arisen. It is very instructive to see how these cells lie near each other in groups of two, three, and four, so that their future destiny is easily perceived. Spots, which before were united in round groups and plates, are seen to send out various branches which attempt to unite, so that a network of cells, in certain spots, comes to view.

3. A *third stage* is often observed, particularly in insane asylums. The formation has now all the characteristics of a membrane. This is of varying thickness, but never very thick (from one-half to one mm.), and is easily torn in large strips from the dura. Usually the epithelium of the dura is torn off with it, and can be seen without much difficulty in scales on the outer surface of the new membrane. The epithelium of the arachnoid is also intact. The membrane has a yellow, in thicker spots a light-brown color. Under the microscope one sees the following appearances, which explain the increased firmness:

The spindle-shaped cells, developed from the white corpuscles, as before mentioned, have united with each other by their prolongations, so that long rows of cells, in clusters of two, three, and four, are found in the rod-like arrangement of the white corpuscles previously spoken of. But between these rows another arrangement is found: a net-like connection of the cells has been formed by the union of their prolongations. This web, with nuclei in the cells, is the commencement of the new connective tissue; the rows are the young capillaries, which contain for the time no blood globules. On the other hand, the retrograde metamorphosis of the lumps of protoplasm, into which the red blood globules have been transformed, is in full progress. The outline disappears more and more; a fine detritus is produced, of unknown chemical composition; a flaky pigment becomes more and more apparent, particularly along the rows of spindle cells, as if it were pushed into line by the prolongations of the cells.

4. A *fourth stage* shows further alterations. The new membrane is now dotted with blood-red spots, appearing like punctiform, and here and there confluent, extravasations—and also proved to be so on microscopical examination. Also complete blood-vessels are found conducting blood. Their lumen is large,

their structure is that of capillaries, but they are larger, as Rindfleisch has well described. At this time the delicate vascular adhesions, which stretch between the neomembrane and the dura, and also, though more rarely, between the membrane and the arachnoid, can be found. These are described by all observers. Of the change of the rows of spindle-cells to blood-vessels, we are not able to give a positive opinion; it would appear that the spindle-cells are transformed into endothelium, and that the plates thus resulting roll themselves together in the form of a tube. Between the vessels, a web of protoplasm with nuclei is found, which must be classed as young connective tissue. Young vessels sprout up from the dura and arachnoid, and unite with those of the new membrane. These changes are in many respects not clear; we will not attempt to decide what rôle the wandering cells play. It is at this stage that the dura is found more injected than usual; but we have never been able to see a perfect injection of the art. meningea med., as described by Kremiansky.

The thin-walled vessels of the new membrane later become surrounded by a more solid coating; a media and adventitia appear; but these complete vessels are always short, and empty into a web of comparatively wide capillaries.

We believe it proved that the new-made vessels are supplied with blood from the neighboring parts. But the connection of the membrane and dura, or arachnoid, is not on that account an intimate one; in by far the majority of cases, on the surface of the pachymeningitic membrane, large flakes of uninjured epithelium were seen, and appearances, such as Rindfleisch represents on page 563 of his Handbuch, were found by us only on limited portions of the new membrane.

The membrane may now assume a stationary condition; it becomes firmer and firmer, and in time poorer in blood-vessels, and finally there remains a thin, clear, and transparent membrane, such as is sometimes found, with various other processes, in the cadaver, without there having been a suspicion of its existence, as the symptoms attending its beginning had passed away long before. Unfortunately this favorable termination only occurs in the minority of cases; the recurrence of extravasations seems to be chiefly due to the delicate and friable construction of the greater number of these membranes.

5. A pseudo-membrane is then found, loosely united with the dura by fibres containing blood-vessels ; this carries on its inner surface a thick or thin layer of blood, which, if arising slowly, presents, in every particular, the appearances described in paragraph 1. The same changes take place—formation of connective tissue and vessels from the characteristically arranged white corpuscles—and soon a new membrane of later date is deposited on the old, becomes connected with the latter, and less constantly also with the arachnoid, by blood-vessels. This can be frequently repeated, the differences in the results being due to the fact that the amount of blood extravasated is not always the same. This may vary in thickness from one millimetre to two centimetres. That organization does not take place in extravasations of the latter size, is easily explained by the fact that death is caused by such an extravasation in the great majority of cases. Many cases of pachymeningitis can be found in the literature of the subject, where a thick clot is found internally, and several membranous layers externally.

6. If the new extravasations do not attain a dangerous thickness, a number of membranous layers are gradually formed one upon another, and between these very frequently smaller and larger clots. There is but a difference in degree between these and an extensive extravasation between two membranes, resembling a sac of blood. If a second interstitial hemorrhage follows the first, and a third also, the series of sacs with bloody contents in different degrees of change is completed. From the pachymeningitis, a hæmatoma has been formed.

7. Not only extravasations of blood occur between the membranes, but also serous transudations ; in confirmation of this, we would cite the well-known case, called by Virchow hydrocephalus externus pachymeningiticus, and also the observation of Guido Weber, who saw, in a laminated hæmatoma, blood in one cavity and yellow-green pus in another.

Localization.—It is a fact, quoted by all observers, and made good use of by some, that the pachymeningitis is more frequently established on the upper part of the brain, along the falx, spreading down the curved portion of the frontal and occipital lobes, and also down the sides towards the fossa Sylvii. Kremiansky found that, in fifty-four out of sixty-five cases, it corresponded exactly in extent to the parietal bones. We must limit the assertion that these boundary-lines are so rarely passed; in the very freshest cases the blood is found to have spread wherever it was possible for it to go. As these cases all occurred in patients suffering from dementia paralytica, blood was found, owing to the great diminution of the brain in the anterior middle cranial fossa, on the upper surface of the tentorium, even on the clivus. These cases, if they are fresh enough, give evidence that the primary exudation, if sufficiently large in amount, will follow the law of gravitation, and sink to the lower parts of the cranial cavity. This only occurs, however, in the minority of cases, for the simple reason that the primary hemorrhage is rarely extensive. We have recently observed a case where a hemorrhage, secondary to, and occurring under, a pseudo-membrane, extended beyond the limits of the latter and reached the base of the cranium. The secondary hemorrhages, which take place in the meshes of the membrane, or between two membranes, have naturally a limited space for spreading. Further, it would be difficult to understand how the middle meningeal artery could be the source of the hemorrhage in those cases where the extravasation is found at the base of the cranium. We cannot as yet explain these cases satisfactorily, but certainly the hemorrhage does not arise from the art. meningea media.

From the carefully described cases to be found in the literature of the subject, fifty-six per cent. embrace the surface of both hemispheres; forty-four per cent., that of one only; these figures correspond not only with my own experience, but also with that of others. It would undoubtedly be interesting to give accurate figures of the different varieties of pachymeningitis; but this is impossible, from the fragmentary and indefinite descriptions too frequently found.

We have not been able to find, among the cases where one side alone was affected, a marked preference of this affection for one side or the other; both seem equally often affected. It would be easy to collect, in searching through the literature of the subject, a considerable number of cases where the extravasation had spread to the base. These will be well known to those who have had opportunity to make many autopsies of persons who had suffered from dementia paralytica. The discrepancy between these observations and those of Kremiansky is due to the fact that he did not collect his material in insane asylums, and consequently did not see great shrinkage of the brain with formation of a large intervening space between the cranium and brain

From the above statement it will be clear that we cannot agree with those who hold that the pachymeningitic product is given off from the dura. Our reasons have been stated above; they are briefly as follows:

a. Although we have had a large material at our disposal, we have been unable to find even a trace of initial inflammation of the dura.

b. We have never seen dilatation of the arteria meningea media spoken of by Kremiansky.

c. The epithelium of the dura remained intact until the formation of vessels in the new membrane, and the establishment of circulation there.

d. The first stage of pachymeningitis is not the formation of connective tissue, but simply an extravasation of blood.

e. This undergoes the changes which usually take place in a coagulum.

f. This develops from itself the succeeding new-formation. It remains for further investigations to determine the function of the wandering cells.

In regard to the formation of the large unilocular or multilocular hæmatomata, we agree with the prevailing view, viz., that the accumulations of blood come from already existing pseudo-membranes. The effects produced within the cavity of the cranium are entirely mechanical.

It is of much importance to learn the changes that take place in other organs, not only those of the skull, but also those in other parts of the body:

a. Changes in the *skull.*—From the apparently conflicting reports (thinning, Hyrtl; thickening, Textor; elevation, Cruveilhier; osteophytes on the inner surface, Rokitansky, Cruveilhier, Textor), it is evident that no constant appearances are found. One must avoid attempting to establish a connection between the pachymeningitis and anomalies or separate affections of the skull, which have no connection whatever with it.

b. Adhesions of the dura.—These are quite inconstant. This condition has, when found with the pachymeningitis, been considered to be in some way connected with it. This we cannot admit. The facts have been stated above.

c. Pia.—Very frequently we find in the pia those changes which accompany atrophy of the brain (œdematous infiltration, slight cloudiness) or those attributed to frequently recurring fluxionary hyperæmia (formation of small fibrous callosities, or at times a thick dense leathery membrane, in connection with atrophy of the brain). So far as the relations of this membrane to the brain are concerned, it will be found at one time easily separable from the cortex, at another, so firmly united as to cause a superficial lesion of the substance when attempts are made to remove it; this depends upon the condition of the surface of the brain, and also upon the processes which have affected the whole of the brain, causing a reduction in its size.

d. Substance of the brain.—The direct effects upon the brain are different according to the thickness of the pachymeningitic organization. The thinnest extravasations and membranes are not sufficient to cause any change of the surface of the brain, especially if any marked atrophy of the brain exists. We have often seen a yellow coloration of the arachnoid, of the subarachnoid fluid, and even of the surface of the brain, penetrating to a greater or less depth; but there were no injuries to the tissues, so that we had the appearances of a post-mortem diffusion before us. The further effects of a large one-sided hæmatoma are those of compression. The corresponding hemisphere is flattened, the greatest depression being usually at the top, or a little outward. The convolutions are flat, the sulci narrowed. The compression is exerted quite deeply; in some cases we have seen the ventricle reduced to half of its size

—even on the base of the brain the pressure is evident by the flattening of the temporal convolutions.

The pressure often produces its effects upon the other hemisphere; the falx is curved towards the healthy side, and the hemisphere compressed laterally from the middle of the brain. In some cases a depression of the tentorium was recognizable, and the evidences of pressure on the cerebellum and medulla oblongata could be demonstrated. The other changes in the brain were only remotely connected with the hæmatoma. It is stated that superficial lacerations of the cortex of the frontal lobes have occurred from a suddenly developed hæmatoma; but a microscopical examination of the compressed cortex has never shown us anything more than anæmia in a high degree, and obliteration of the cavity of the vessels; solutions of continuity and lacerations we have never seen.

Independently of hæmatoma, various changes are found, especially atrophy and the different forms of contraction of the brain. Without doubt, the decrease in size and weight of the brain of drunkards is one of the important factors, varied greatly by various modifying influences. Senile atrophy also often plays an important part, and in a number of the reports hydrocephalus internus is mentioned. Further, we frequently find that form of atrophy which usually follows general degeneration of the cerebral vessels. Atheroma of the basilar arteries, fatty degeneration and calcification of the intracerebral arteries are often mentioned, accompanied, it is true, by general atheroma. In some cases descriptions are given which indicate a diffuse sclerosis and a decrease in volume. Finally, among the records we find mention made of a large number of cases of dementia paralytica. In fact, it appears (accurate figures are not possible) that pachymeningitis more frequently accompanies that form of chronic inflammation of the brain, the symptoms of which we class under the general head of dementia paralytica, than it does alcoholic atrophy of the brain. Of further affections of the brain, the following are mentioned: necrotic softening (arterial thrombosis), apoplexies in the large ganglia. Guido Weber reports a case of soft sarcoma of the dura.

We have met with pachymeningitis in no disease so frequently as in dementia paralytica; we have also encountered it in chronic melancholia, progressing to dementia, with contraction of the brain, and varied senile changes. In a few cases it occurred with tumors of the brain Considering all the changes, we are forced to the conclusion that the great majority of cases of pachymeningitis occur with changes which cause a reduction of the volume of the brain. May not all cases be considered as so occurring? Although we have never observed a case where the coincidence was not present, yet the area of observation of an individual is too limited, and the statements in the literature of the subject too indefinite, to establish a positive conclusion in regard to this. The question must therefore remain for the present undecided.

e. Lungs.—All possible affections of the lungs have been found with pachymeningitis. Various observers have found it in connection with acute diseases; miliary tuberculosis, pneumonia, pleuro-pneumonia, pneumo-thorax are mentioned; further, phthisis, emphysema have been mentioned—conditions which have a certain significance in connection with the appearances of the brain.

f. Heart.—Kremiansky was the first to call attention to the connection between pachymeningitis and affections of the heart. Singularly enough, the founder of that theory which offers as the cause of this affection the fluxion of the arteria meningea media, mentions insufficiency of the tricuspid as the predisposing influence! Guido Weber has published other articles, and it is interesting to see in these, also, how pachymeningitis occurs in processes causing an increase of the blood pressure in the venous system. Pericarditis also (Hasse) is mentioned as a cause.

g. Kidneys.—Bright's disease is often reported, and cirrhosis of the kidney several times. Mention is also made of hydronephrosis with multiple renal abscesses.

h. Stomach and liver.—In this connection belongs carcinoma, which during the terminal marasmus becomes complicated with pachymeningitis. We would also mention the carcinomatous dyscrasia in general.

i. Intestines.—These are often found tuberculous; but this affection is only one symptom of a general tuberculosis, seated chiefly in the lungs.

k. Alterations in *the blood* play, without doubt, an important part in the origin of hæmatoma. We have seen it complicated with leukæmia, with anæmia splenica, but, more than all, with anæmia perniciosa. These, as is well known, all predispose to hemorrhage; so that the existence of a hemorrhagic diathesis has been asserted.

l. Pachymeningitis has been observed with *puerperal disease.*

m. It has been seen with *typhus*, and with *relapsing fever* (*febris recurrens*); according to Kremiansky, very frequently with the latter.

n. *Variola* is said by Hasse to have a predisposing influence; *scarlatina* is also mentioned, but not so positively.

o. *Acute articular rheumatism* is spoken of by Lancereaux and Hasse; but this is considered doubtful by Kremiansky.

p. Finally, in a number of cases, there were *injuries of different kinds*, causing a solution of continuity of the dura, the walls of the sinuses and the like. The subject will be again spoken of; we state simply that the flat extravasations, caused by injuries, have appeared to all observers, whatever their views in regard to the origin of pachymeningitis were, as an ordinary pachymeningitis.

Etiology.

Since we cannot accept the theory of the inflammatory development of pachymeningitis from the dura, certain questions must be answered, if a clear understanding of its etiology is to be obtained.

Where does the hemorrhage arise? In the above statements it will be seen that the first manifestation of the disease was an extravasation of blood. The earlier French writers have often proposed the above question without finding a satisfactory answer. It is of course easy to point to extravasations following an injury (nail in the dura [Guido Weber], laceration of the dura in fractures of the brain), and in these cases there is no need to cudgel one's brains for an explanation. If no source for the hemorrhage was manifest, a "bloody exhalation," without injury of the vessels, or capillary bleeding, was suggested. Durand-Fardel speaks of a case where a cerebral apoplexy burst through the arachnoid, causing a clot between the dura and the arachnoid. These cases are, however, very rare.

Our observations have not enabled us to find the source of bleeding in all cases, partly because attention was not at first paid to this point. In some cases of later date the source was ascertained. In regard to this matter we would call attention to the following facts :

a. In some cases the greatest thickness of the extravasation is not found at a point corresponding to the arched portion of the os parietale, but on the falx along the sinus longitudinalis. The veins, leading from the surface of the brain to the sinus, deserve more attention than has hitherto been given to them. They are often enlarged and varicose, the walls thinned, and therefore easily ruptured. This condition often coincides with atrophy of the brain, before spoken of, in which, as is well known, the tendency to a vacuum is compensated for by an increase in the serous fluid in the meshes of the pia and in the cerebral cavities. Without doubt, the contents of the above-mentioned vessels are also influenced; the vessels are found very full, even when there is no marked congestion elsewhere. This persistent increased fullness must have an effect upon the varicosities, especially in view of the condition now about to be described.

b. We have found the above-mentioned veins fattily degenerated in dementia paralytica, in encephalomalacia senilis, and in atrophy of the brain in old age. Since we have directed our attention to this matter, material from other diseases has not yet been presented us, and we are therefore unable to generalize.

c. We have often found in these enlarged veins, with degenerated walls, an old thrombus, extending to the sinus longitudinalis.

d. Finally, in one case, where a vein, just before its entrance into the sinus, contained a thrombus, a rupture was found in the peripheral part (in encephalomalacia senilis). The consequence was an extravasation of blood, half a millimetre thick, spread over the bregmatic region.

e. Attention should be directed to the vessels of the Pacchionian granulations of the arachnoid. The frequent venous congestion of these parts is well known; but we have never been able as yet to find a spot where a rupture had occurred; old thromboses, however, were frequently seen in the veins.

f. How far the vessels of the pia may be involved we would not venture to state. They are frequently degenerated in atrophy of the brain. In some cases we saw subarachnoid extravasation (emanating from these vessels) connected with fresh pachymeningitis; in one case the arachnoid was ruptured, and the effusion of blood above the membrane was continuous with that below it; it was uncertain, however, whether the extravasation between the dura and arachnoid came from a subarachnoid bleeding or not.

This is all that has been observed—during a rather short time, it is true—in regard to rupture of vessels.

It is clear that a small bleeding can only extend from the source of hemorrhage over a limited area; we have shown that a copious hemorrhage spreads further, and can, under certain circumstances, reach the lowest parts of the cranial cavity. The limitation of the first bleeding depends upon its limited amount, the localization upon the place of rupture of a vessel. The limitation of the subsequent hemorrhages comes from the confined locality in which they usually occur. They are rarely able to extend beyond the boundaries of the membrane already formed. This only occurs when the bleeding comes from the free under-surface of the new membrane, which, as has before been mentioned, is occasionally the case.

To come to a second question: Why does hemorrhage occur in only a limited number of cases, although degeneration, varicosity, and distention of the vessels, as before mentioned, are frequently seen?

First, the question is to be answered, do pachymeningitic changes occur in perfectly healthy persons? After a careful examination of all cases described, and from personal observation, the conclusion arrived at is, that pachymeningitis does not occur in perfect health—except in case of an injury. We were unable to find a single case sufficiently reliable to overthrow this opinion.

We must therefore turn to the general conditions under which pachymeningitis is observed; and here we again call attention to the coincidence with atrophy of the brain, before alluded to. We are aware, however, that from the facts observed a generalization universally applicable cannot be made.

1. Pachymeningitis and hæmatoma have been frequently observed *together with affections of the lungs:* tuberculosis with pyopneumothorax, pleurisy alone, emphysema with bronchiectasis, pneumonia. It is noteworthy that in the majority of cases the affections were of the following nature: chronic diseases of long standing, diseases which impair seriously the constitution and nutrition, and, finally, diseases which one and all are associated with congestion of the venous system. All the affections of this category are accompanied by cough.

2. A certain number of the cases of pachymeningitis and hæmatoma were associated *with affections of the heart and vessels.* Mitral, aortic, tricuspid lesions, and pericarditis are mentioned. In many cases widespread atheroma is spoken of. Venous stasis is to be noted in this connection, and also the cachexia which eventually follows degeneration of the heart and blood-vessels; the connection between degeneration of the vessels of the brain and its membranes and the shrinkage of the brain should also not be forgotten.

3. The relative frequency of diseases of the kidney is remarkable: *morbus Brightii* and *cirrhosis.* Such a case came under our observation, characterized by various exudations, hydrothorax, and ascites. The brain was atrophied, all the arteries atheromatous, the venous stasis extreme.

4. Of the greatest interest in regard to the etiology are the *diseases of the blood*, to which pachymeningitis is added. *Pernicious anæmia* is the most important; in one-third of the cases, hæmatoma occurs as a complication. It was in this disease that we found (in a few cases) the changes in the very first stage; but no trace of an inflammatory affection of the dura, no sign of fullness of the arteria meningea media could be seen, although in

the great poverty of blood of all parts, this would have been recognizable with peculiar distinctness, had it been present. The appearances in these cases, however, could not be distinguished, either microscopically or macroscopically, from what is presented by the hæmatoma in dementia paralytica or the dementia of drinkers. It is to be remembered that, in pernicious anæmia, other hemorrhages, partly explicable and partly not, are liable to occur (in the brain, in the skin, in the retina, under the serous membranes, in the stomach, intestines, in the mucous membranes), but no one has ever thought to refer these to an inflammatory origin. In all cases degeneration of the blood-vessels is not found; the bleeding, too, between the dura and the arachnoid has not yet been explained; but certainly no form of pachymeningitis speaks so strongly against an inflammatory genesis as that which sometimes accompanies pernicious anæmia.

Pachymeningitis occurs in *scurvy;* here, however, we have no personal observations to offer. We have seen it in a case of *leukæmia*, in which it occurred during the hemorrhagic diathesis; here also, although the coagulated blood was quite recent —only as thick as paper—no trace of inflammation of the dura could be found. An interesting observation of Walder belongs under this category; he saw the affection occur in a case of *hæmophilia;* the anæmia of the dura is expressly mentioned, and the theory of a primary extravasation offered as an explanation. Finally, many cases exist where *syphilis* was supposed to be the original disease; the connection is not clear; certainly no one will be disposed to refer the affection to Heubner's disease of the cerebral vessels.

5. It is an interesting fact that pachymeningitis can develop during the paroxysms of *pertussis* (Wilks); here the high degree of passive venous congestion is to be remembered.

6. *Typhus* [*abdominalis*] *and febris recurrens.*—Typhus is mentioned by some early French writers; recurrens by Kremiansky. But the latter denies any genetic connection with the disease, and considers that the patients were drinkers. But the occurrence of hemorrhages between the dura and the arachnoid in typhus, cannot be doubted. Griesinger and Buhl describe

undoubted cases; the first, however, adds that it was simply a hemorrhage, and not a pachymeningitis. After what we have said above, it will not seem strange if we class these as cases of pachymeningitis. Several occurred in the third week of the typhus, others in the subsequent marasmus. We ourselves saw a case, where, in the tenth week, such a bleeding, added to extensive bronchiectases arising during typhus, caused death. The brain was atrophied, the vessels were not examined. If the bleeding occurs in the first stage of the typhus, we are unable to give an explanation; if during the marasmus, the same influences spoken of above will apply here. [By typhus, typhus abdominalis or typhoid is clearly meant.—TRANS.]

7. *Acute rheumatism*, mentioned by Lancereaux, denied by Kremiansky, again mentioned by Hasse. We have not seen anything resembling this in either the acute or protracted form of cerebral rheumatism.

8. *Variola, scarlatina.*—We have no right to deny Hasse's statement in regard to variola and scarlatina; but at present we are unable to explain the connection.

9. *Chronic alcoholism.*—According to Lancereaux, the most frequent and important cause. Kremiansky gives even more importance to it. They differ, however, in their explanations. While Lancereaux (relying on the experiments of Lallemand and Perrin) believes that alcohol is exhaled from all surfaces, and acts as an irritant, and that this occurs also on the surface of the dura, Kremiansky ascribes his "constant hyperæmia of the middle meningeal artery" to the alcohol. Kremiansky endeavors to support his theory by a series of experiments on dogs fed with alcohol. We, too, as will be imagined from what has been said above, consider that chronic alcoholism is a fruitful source of this affection, but for a different reason. We believe the atrophy of the brain, with degeneration of the vessels, to be the main cause; this occurs in different individuals at different times, but is certain to result in time. Transitory, but frequently repeated stases, will cause rupture of some vessel.

10. *Affections of the brain.*—Many of these have already been spoken of. Pachymeningitis is found in the terminal stage of apoplexies and necrotic softenings of every nature, and also in

that of tumors; but the fact is to be noticed that towards the end of life an atrophy of the brain occurs in all these diseases, especially when an extensive atheroma lies at the bottom of all these affections. Further, the disease in question is found in all forms of atrophy of the brain, which are accompanied by œdema of the pia and hydrocephalus internus caused by the atrophy. Here, again, atheroma is an important factor, and also senile involution of the brain, and finally alcoholism, which causes an atrophy of the brain, before old age has produced any effect. But the disease is nowhere so frequent as in the chronic psychoses, especially those which cause the greatest degree of atrophy; and here we should particularly mention dementia paralytica, due to chronic inflammation of the brain. Pachymeningitis also occurs in marantic dementia, especially after atrophy begins, and the primary stage of the psychosis is passed.

11. *Local causes.*—Injuries are important. Schneider's statistics (Inaug. Diss., Zurich, 1874) show that injuries occurred in seventeen out of seventy-four cases. We can almost believe that these would appear even more important as causes, especially in affections of the brain, if statistical data were given. Kindt found a connection between othæmatoma and pachymeningitis which is interesting, as the former (Gudden) only comes after injuries. These are blows, falls, also foreign bodies, and fracture with laceration of the dura, tearing off the wall of a sinus. Neoplasms, gummata, sarcomata, fibromata of the dura, should not be forgotten here.

It would be desirable to show that all these states tend to cause rupture of the vessels in the immediate neighborhood of the dura and arachnoid. This is, however, at present only in a small measure possible. Still less, however, can it be shown that the above conditions favor an arterial fluxion of the arteria mening. med., a fluxion, moreover, which but very few have observed.

In chronic affections of the lungs, if the accompanying conditions are favorable (atrophy of the brain, degeneration of the vessels), the hemorrhage can be explained as arising from the vessels before spoken of, while they are in the condition of passive congestion which the attacks of severe coughing produce.

The coincidence of pachymeningitis with pneumonia is easily explicable if these patients be drunkards.

The occurrence of the hemorrhages in diseases of the heart, particularly those combined with extensive degeneration of the vessels, is not strange; and the same is true of their occurrence in the course of diseases of the kidneys, which indirectly cause the same changes. The above-mentioned blood diseases promote a tendency to the rupture of vessels, as shown by the frequent hemorrhages in leukæmia, scorbutus, pernicious anæmia, and hæmophilia.

All that is necessary has already been said in regard to alcoholism and affections of the brain. We will only add, that Kremiansky's experiments of feeding with alcohol are not the only ones that have been made in regard to this point. Ruge has repeated these, and found in no case hyperæmia of the arteria meningea media, or evidence of pachymeningitis. Finally, Sperling's experiments should be mentioned; he injected fresh blood from rabbits under the dura of the same animals, and so produced a pseudo-membrane, which could not be distinguished from that observed in pachymeningitis. Injections of irritants, however, never caused a pachymeningitic inflammation, but simply an ordinary suppuration. Defibrinated blood did not give rise to the formation of a pseudo-membrane. The fibrine, therefore, seems to play a part, which our observations do not as yet explain.

From all these considerations we must conclude, that, viewed from the etiological standpoint, the theory of a primary inflammation of the dura is not tenable. It is both desirable and necessary that the observations above made, and the conclusions drawn from them, should either be confirmed or refuted by future experiments.

All authors point to the fact that pachymeningitis is *a disease of old age;* this would also be supposed from a consideration of the etiological facts. A few cases have been observed in children (child of eight years, and one of two years and three-quarters, Wagner; one between three and four, Steffen; one of seven months, Moses; one of six months, G. Weber). We are entirely unable to give a satisfactory explanation of these cases.

The following table has been constructed from all the cases of which we were able to gain any knowledge:

Under 1 year	2.7	per cent.	of all cases.
From 1 to 10 years	2.7	"	"
" 10 to 20 "	1.5	"	"
" 20 to 30 "	5.5	"	"
" 30 to 40 "	12.5	"	"
" 40 to 50 "	17.6	"	"
" 50 to 60 "	13.5	"	"
" 60 to 70 "	19	"	"
" 70 to 80 "	22	"	"
Over 80 "	3	"	"

According to the statement of Durand-Fardel, 77.4 per cent. of all cases were men, and 22.6 per cent. women.

Symptoms.

There are great obstacles in the way of making a reliable statement of the symptoms, and also in establishing an accurate basis for the diagnosis. The symptoms vary as in no other affection of the brain, although the anatomical appearances are quite uniform. This is easily comprehended if one considers:

1. That the hæmatoma is a complex disease, composed of various processes, which may be limited at different stages. The following anatomical and clinical varieties are to be considered:

a. The first hemorrhage may be so copious as to cause death. This is characterized by a short apoplectiform course, hardly recognizable.

b. The initial hemorrhage is not large, but lasts some time, prolonging the symptoms, which may be slight at first, but become more severe afterwards, and cause death.

c. The initial bleeding is slight or moderate: membranes are formed from this; no new hemorrhages occur; the new organization shrinks; the primary symptoms will vary in intensity; the patient recovers with comparatively good health.

d. A primary hemorrhage occurs, developing later into membranes; there is pause for a time, and then come new hemorrhages, causing a sudden or lingering death. The course is characterized by two acute episodes separated by a chronic stage.

e. The second hemorrhage does not cause death, but a third or a fourth one occurs. A number of acute attacks follow each other, separated by periods more or less free from symptoms.

We think a diagnosis is rarely possible in the two first varieties; in the third the disease is rather more readily recognized; but in the last two there are no serious reasons why many cases should not be diagnosticated.

2. That a disease lies at the bottom of this affection, and that its symptoms must first be separately recognized. Since in many cases brain symptoms existed previously (atrophy of the brain, dementia paralytica), a diagnosis is often impossible.

3. That the localization varies. The disease may be on one side, and remain so; or it may extend over both sides in the beginning; or it may commence on one side and afterwards extend to the other.

4. That the effect of the hemorrhage depends upon the degree of the cerebral atrophy; for example, that which hardly causes headache in a demented paralytic, may lead to far more serious disturbances in a drunkard whose cerebral atrophy has not advanced so far.

5. That, in accordance with our views of the origin of the hæmatoma, uniformity in the extravasation is not to be expected any more than uniformity in the rapidity of the hemorrhage.

If we follow the plan which simply places opposite the number of cases the symptoms observed in individual cases, no satisfactory result would be reached. The anatomical processes being different in different cases, we are compelled to give several separate pictures of the disease; therefore, any consideration of the general course becomes unnecessary.

I. The hemorrhage is so severe from the beginning, that death occurs soon or immediately. In these cases, the manifestations of cerebral compression, coming on suddenly and rapidly increasing to a very marked degree, are observed. These can scarcely follow from any other cause than a copious hemorrhage. There is, however, the greatest difficulty in deciding whether the extravasation is intra- or extra-cerebral.

3. A woman, sixty-three years old, of good constitution, suffered from an old hemiplegia (the variety not mentioned); suffered also from congestion of the brain from time to time. One day she experienced a violent mental excitement. Two days afterwards she suddenly lost consciousness; the face became red; the temporals pulsated violently; respiration became stertorous, short, and difficult. Full pulse, complete loss of sensation and motion in both extremities. Death in forty-eight hours. *Autopsy.*—Enormous quantity of black coagulated blood in the arachnoid cavity over the left hemisphere. This portion of the brain is much compressed by the blood which has extended to the base of the brain, and even to the cerebellum (Rostan).

There are many such cases to be found in literature. Hitherto they have not been classed as pachymeningitis; it is our opinion, however, that they should be so classed. The following additional symptoms have been observed in similar cases: severe headache, just before the attack; after loss of consciousness had occurred, contracted pupils, not reacting (in one very acute case, observed by myself during the whole course, the pupil failed to react, and was moderately dilated); in a few cases, paralysis of the facial nerve on the side opposite to that of the hæmatoma; sometimes hemiplegia. These latter symptoms only occur in one-sided hemorrhages. A marked change in the color of the face is another of the symptoms reported; at the commencement of the attack, which is usually sudden, the face becomes flushed; the pulse is full and rapid, but soon becomes small and irregular, with pallor of the countenance. In some cases the pulse is slow; in others there is an increase in rapidity, continuing up to the time of death. Contractures of the extremities and slight transitory twitchings, were present in a few cases.

The question, whether a diagnosis is possible in the majority of the cases, must be answered in the negative. There are many intra-cerebral hemorrhages, which cause great compression by their size; deprive the cortex of its functions, and perhaps manifest themselves by the weakness of one side of the body, followed soon by a similar paralysis of the other side; give rise to some symptoms of irritation, transitory contractions, or convulsions of limited extent; paralyze the centres governing the motions of the iris and bulb, so that the muscular apparatus is left to its own elasticity; and finally impair the vital centres situated in the medulla oblongata in such a way that their functions are either imperfectly performed or entirely lost.

A diagnosis is perhaps possible in those cases where the extra-cerebral hemorrhage causes symptoms pointing to a lesion first of one, then of the other hemisphere—a matter to be spoken of later. One symptom is not precisely the same in both forms: in extra-cerebral hemorrhage the narrow and fixed pupil occurs at the beginning, when unconsciousness has already set in, in a greater number of cases than in intra-cerebral hemorrhage—a symptom which, in connection with the impairment of the sensorium, points more to injury upon the surface than to one in the substance of the brain (Griesinger). But any one, who has had experience in the relative worth of such symptoms, will not be very sanguine as to their value.

In a small number of cases the extravasations on one side cause severe convulsive movements of the opposite side; a still smaller number are characterized by short epileptiform convulsions. The question arises, why all cases of such a characteristic superficial lesion do not produce violent convulsions. This must depend on certain modifications in the intensity of the compression of the cortex. Every irritation of the surface does not give rise to convulsions; a continuous even pressure is less likely to produce them than are a number of isolated irritations. Further, the period during which convulsions occur is a very brief one; the pressure quickly increases to such a degree that all irritability ceases. Finally, it is very questionable whether direct irritants cause convulsions; more probably they depend on transitory alterations of the circulation (contractions of the vessels); in the affection under consideration only a very limited effect could result, as the possibility of variations in the size and capacity of the vessels would be removed quickly by the rapid increase in the amount of pressure.

II. Unlike the above, very many cases are reported where extravasations were found after death, though there had been no suspicion of their existence during life. It is hardly necessary to cite examples; in the record books of insane asylums these so-called pachymeningitic processes, fresh and more developed, are repeatedly mentioned among the post-mortem appearances, without any other than the ordinary symptoms of dementia paralytica having been observed during the patient's lifetime. We cannot coincide with the opinion that the apoplectic and epileptic attacks of paralytics have any connection with the pachymeningitic process. The absence of symptoms in these cases is due to the great atrophy of the brain, which allows the development of a comparatively large hemorrhage without necessitating any great compression. These hemorrhages, however, are rarely large.

In other varieties of disease of the brain fresh hemorrhages occur now and then, with symptoms so various that, in any individual case, we cannot safely draw any inferences from their occurrence. We have seen, in old patients suffering from melancholia agitans, pachymeningitis occur, causing immediately a change in the condition of the patient; the jactitation ceased, and drowsiness, with severe cephalalgia, set in. But in such cases we never saw symptoms of such a definite character—such as hemiplegia, or characteristic alterations of the muscles of the eye, etc.—as to justify one in expressing an opinion in regard to the nature and locality of the lesion. At the same time it cannot be denied that theoretically such symptoms may occur, and may in isolated cases enable us to form a diagnosis.

III. The hemorrhage at first is slight, but does not cease, and leads to severe symptoms and death. This class of cases is somewhat more easily understood

4. Drinker, sixty-three years old; slightly demented for some time; memory and judgment impaired; atheroma, chronic bronchitis. Complained one day of headache and dizziness; his face became flushed and hot; full hard pulse, 106; throbbing of blood-vessels, elevated temperature; slept a good deal, could be awakened only with difficulty; stertorous breathing during sleep; slow and helpless in all motions. (First day.) Sleepiness gave way to unconsciousness, could be awakened only with great difficulty; patient complained of severe headache; there was fever; vomiting, no constipation; quick pulse, 100; external signs of congestion of head; heat, flushing; pupils contracted, but reacting normally; no paralysis of muscles of the eye; sensation normal; no paralysis of extremities; increased reflex activity of whole body. (Second day.) Patient worse; sopor; slower pulse, 90, very full; fever; vomiting once; normal dejection; left facial paretic; narrow pupils with impaired reaction; no paralysis of extremities, but sensibility can no longer be satisfactorily tested; increased reflex activity. Slight delirium at times, when sopor

is less marked. (Third day.) Sopor; pulse 70, full; patient cannot be awakened; both pupils dilated, left more than right; no reaction; left facial paretic; many confused movements; kicking and tossing about of the extremities, but movements of left arm and leg less active than those of the right; left extremity weaker; nothing definite about the hypoglossus. (Fourth day.) Deep coma on the following day; reflex activity diminished; no movements; paralysis on left side still evident on passive motion; paralysis of left facial, doubtful in regard to hypoglossus; slight twitching in both extremities; pupils more dilated than on previous day, left somewhat wider than right, no reaction; pulse 120; irregular fever; death on the fifth day. *Autopsy.*—Fresh hemorrhage on both sides; no membrane; greatest thickness of the extravasated blood on the right side, where it measures one centimetre, while on the left it measures but three-quarters of a centimetre; it extends to the base, becoming gradually thinner; it is thickest along the falx; blood in greater part coagulated, but partly fluid; atrophy of the brain; hydrocephalus internus in spite of the moderate compression of both hemispheres. (Case of my own.

Similar cases are mentioned, particularly by the French writers. In the beginning the chief symptom was an intense cephalalgia (with or without vertigo) in almost all cases; after this, drowsiness, increasing gradually to sopor, and finally to coma. Fever of varying intensity was also present. None of these are characteristic of the process under consideration. They indicate simply a commencing morbid process on the surface of the brain,—one which might perfectly well start from the pia, or even from a previously latent process situated more deeply in the substance of the brain. The increasing feebleness of the pulse, during the transition to coma, also furnishes no further light. Often more clearly defined symptoms appear; of these contraction of the pupils is the most important; this manifests itself at a very early stage and remains constant for a short time, after which the reaction takes place slowly. In some cases the pupils are unchanged; in the majority of cases, however, the pupils dilate gradually to a moderate degree, to remain so, not responding to irritation. The greater amount of the extravasation on one side sometimes has the effect of dilating the opposite pupil more than the other. In none of these cases do I find mention made of strabismus. Very important are the gradually appearing symptoms of superficial lesions of both hemispheres, facial paresis, hemiparesis on the same side, and then symptoms of irritation or paralysis on the opposite side—a not very rare coincidence. In one of our cases, where the consciousness remained unaffected a long time, or at least was but partially affected, all the extremities were finally paralyzed. The temperature and pulse curves usually confirm the belief that a severe superficial lesion of the brain is present.

One easily sees that symptoms, like those described above, can be furnished by any meningeal affection, especially as the characteristic paralyses do not occur with uniform prominence in all cases. Among the various groups of symptoms presented by tubercular meningitis, some will easily be found which conform almost exactly to those just mentioned. One would perhaps be disposed to consider as distinctively diagnostic the absence of certain symptoms which denote an affection of the base (absence of paralysis of the oculomotorius and abducens, absence of strabismus, absence of ptosis). But in our experience this will not suffice, as in tubercular meningitis the exudation at the base may be slight, while the affection of the convexity is severe. Contraction of the pupil at an early stage is considered by some (Griesinger) as characteristic; this, however, occurs occasionally in tubercular meningitis. In general the changes in the state of the pupil are much the same in the two affections. Nor can much stress be laid upon the diagnostic worth of contractions of the neck or a sunken condition of the abdomen. We cannot consider either of them as always present in tubercular meningitis. At the same time we must confess that we have never observed these symptoms in a well-marked degree in a case of hæmatoma. In establishing a diagnosis, therefore, the peculiar conditions under which the disease is developed must be duly considered. A hæmatoma is more probable in old persons, where atrophy of the brain can be suspected, arising from any of the usual causes—alcohol, atheroma, old age, chronic psychosis. The chances would favor tubercular meningitis where there was suspicion of tubercular disease in any form. There are many opportunities of error, however. Tubercular meningitis occurs occasionally in middle life; its course may be modified in a great variety of ways; it may occur in persons suffering from atrophy of the brain; and finally the patient's own account of his case cannot be fully trusted. The conclusion, therefore, is that the cases must be very well marked to render a diagnosis possible.

IV. An extravasation gives rise to acute symptoms; a membrane is formed, which undergoes secondary changes; there are no new hemorrhages. A return to a relatively normal condition follows. That such cases sometimes occur can easily be demonstrated by post-mortem examinations; healed hæmatomata, *i. e.*, membranes, are quite often found at the autopsy of persons who have suffered from some form of chronic psychosis, or from dementia. We could quote several such cases, similar to those described by Griesinger;[1] but diagnoses of hæmatoma are always open to doubt if recovery occurs.

As we have no records of our own where an autopsy was made in such a case of healed hæmatoma, we prefer to quote the well-known case of Bouillon-Lagrange, where, after recovery, the existence of the affection was proved.

5. A man, seventy-five years of age, presented symptoms of gradual diminution of all his mental faculties, especially impairment of memory, somnolence, and disinclination to activity. After a fall from a horse, an immediate increase in all the symptoms followed, though no external injury could be found. Headache, rapid decrease of all his mental powers, constant somnolence, extreme forgetfulness, stuttering, after a while paralysis of the tongue and paralysis of the right side, all ensued. This lasted two months, after which there was drowsiness, facial paresis of the right side, deafness, headache, constipation; pulse 48 to 50. The pupils were normal; tongue straight; sensation and motion normal on the left side, and normal sensation on right side, but almost complete loss of motion; the fingers moved with difficulty; dragging of left leg in walking. After this the patient became rapidly worse: coma; almost total general paralysis; stertorous respiration; involuntary discharges; loss of speech; difficulty in swallowing; subsultus tendinum of left arm and leg.

After nearly ten days, an improvement in all the psychical functions; patient begins to speak; is at times conscious. After fifteen days, recollection returns: speech much improved. After twenty days, paralysis improves, at first in one leg. In a month patient can walk, and can move the paralyzed arm; and in a month and a half, but slight impairment in limbs. At the end of four months after the beginning of the attack, patient had entirely recovered, and remained perfectly well for six months, when he was murdered. *Autopsy.*—Fresh fissure of cranium; both sheaths of the arachnoid adherent to the surface of the brain; thickening of the visceral layer of the arachnoid; the same on the right side; but a cyst is noticeable in the arachnoideal cavity, covering the posterior two-thirds of the right hemisphere, causing a marked depression. The cyst is oval; the greatest diameter runs antero-posteriorly; the contents, three or four spoonfuls of a bloody fluid; cyst adherent to the visceral layer of the arachnoid; pia filled with black blood; over the right anterior lobe a small clump of coagulated blood. (Bouillon-Lagrange quotes this case as a proof of the existence of a *paralysie non-croisée.* The fresh extravasation on the right side was probably the effect of the injury which caused death.)

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[1] Archiv der Heilkunde. 1862.

Such cases prove that a hæmatoma is not necessarily fatal. Quite a number of reports of recovery can be found in the literature of the subject.

V. A diagnosis of the affection is more easily made if two attacks can be observed, separated by a longer or shorter interval of relative health. The attacks may be characterized as apoplectiform; the second causes death. In these cases unilocular or multilocular sacs are found on one or both sides; one of the subordinate sacs always contains a considerable effusion of blood, causing death by compression.

The diagnosis is here often not difficult; but it must be borne in mind that in diseases of the brain it is not single symptoms which lead to the establishment of a diagnosis, but rather their combination.

6. Engineer; thirty-one years of age; hard drinker; two years previously, apoplectic attack, preceded by violent headache; felt dizzy and fell; lost consciousness in the course of a few minutes; slight convulsions on the right side, which soon ceased; during twenty-four hours lay in an unconscious state, feverish, with full, slow pulse and pupils contracted, but reacting; during this time a facial paresis of the right side was developed. At the end of the twenty-four hours consciousness slowly returned, but the headache and dizziness continued; the fever disappeared; and at the end of a few days the patient was again able to get out of bed. A period of comparative health followed (one and a half years). During this time patient, however, suffered frequently from headache; the attacks were severe, but short, and during the intervals there was never absolute freedom from pain. A constant diminution of intelligence noticed; impairment of memory and judgment. Patient acts strangely; loses his situation, and drinks immoderately (especially brandy).

One and a half years later, a new attack. After slight condition of malaise, with a few symptoms of delirium tremens, a sudden attack of vertigo occurred.

Violent headache; fever; flushed face; impairment of consciousness; violent delirium, but not that resembling delirium tremens. There was slight sopor after a short time; patient, however, easily awakened, and answers intelligently; pupils both equally contracted, but reacting; no paralysis of facial or hypoglossus; all the extremities weak, but still able to move; sensation normal; no difference in the power of the extremities of the two sides. (First day.) Headache continues in spite of antiphlogistic treatment; patient awakened with difficulty; pupils contracted, the same on both sides, reacting; snoring respiration. The right corner of the mouth droops; no change in the eyelids or hypoglossus; slight convulsive movements in right arm and right leg; slight impairment of sensation of the whole right side; no change on the left side; high fever; frequent pulse; vomiting. By evening, more decided paralysis of the right side; convulsive movements on the left. (Second day.) Sopor; paralysis of the right side; paresis of left; slight contracture of the upper arm; the right pupil dilated, no reaction; the left pupil contracted, no reaction; the right facial paralysis increased; none on the left side; the tongue drawn a little towards the right; high fever; stertorous respiration; a slow, full, and rather irregular pulse. (Third day.) Profound coma; slow, stertorous respiration, with occasional omissions; both pupils moderately dilated, motionless; right facial paralyzed; hypoglossus uncertain; extremities as on previous day; involuntary discharges; high fever; pulse slow, irregular. (Fourth day.) Death after the fourth day.

Autopsy.—Hæmatoma on both hemispheres—one and a half centimetres thick on the left side, one centimetre on the right. Each sac is enclosed by two tough membranes, firmly adherent to the dura, loosely adherent to the arachnoid; three secondary cavities on the left side, two on the right; masses of coagulated blood and some fluid blood in both sacs. (No microscopical examination.) Brain compressed on both sides, more so on the left; great dilatation of the ventricles; hydrocephalus internus; atrophy of the brain.

Similar cases are mentioned by Wagner,[1] Charcot and Vulpian,[2] Bouillon-Lagrange,[3] and others. Such a sequence of symptoms certainly presents a very characteristic picture of disease. A similar chain of symptoms is seen in abscess of the brain; but in this the etiology is different, and convulsions are more frequent. In the above-mentioned case, which is similar to many others, a diffuse affection of the surface of the brain, causing compression, would be immediately suspected. This subsides and then remains latent, leaving traces, however, which would suggest hæmatoma. The second attack is also caused by a diffuse affection of the surface, spreading from one side to the other, and causing paralyses on both sides, and death.

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[1] Jahrbuch d. Kinderheilkunde. I.

[2] Gaz. Hebd. 1860.

[3] Arch. Gen. 1847.

The Separate Symptoms.

a. Headache is one of the most constant symptoms; while it varies in intensity, it is yet never entirely absent. There is throbbing and a sense of pressure in the head; and the pain is said to be as if something moved about in the head (Griesinger). Sometimes it is more violent on the side of the larger hæmatoma; but this varies. It is never accurately localized; and as soon as a new hemorrhage occurs, there is a very decided exacerbation of pain. If, however, the atrophy of the brain is great, a hæmatoma may even attain to considerable size, without causing great headache. This is often found to be the case in patients with dementia paralytica, and in old persons with atrophied brains. Under such circumstances we have known a hæmatoma to form, half a centimetre in thickness, without causing headache; while in other cases, where the shrinking of the brain was slight, a very thin layer of blood caused violent pain.

b. Disturbances of the sensorium and of the intelligence occur in a large number of cases. Of these disturbances some are due to the fundamental disease (alcoholism, dementia paralytica, atrophy of the brain from degeneration of the vessels), while others are of a more acute nature, caused by the hemorrhage. In the beginning, symptoms of cerebral irritation are prominent, but these soon give place to those of compression. The initial excitement, marked with great irritability, sensitiveness to light, ringing in ears, etc., gives place to sopor, and finally to entire loss of function of the cortex of the brain. In other cases the disease begins like a regular attack of apoplexy, characterized by a sudden loss of consciousness, and occasionally followed by death. During the intervals an important symptom is the drowsiness, noticed in a number of cases. Griesinger has called attention to its occurrence, especially in connection with contracted pupils and chronic headache. The coincidence of these symptoms in the course of the disease would appear to have diagnostic value, but it is not always found in all the acute episodes; on the contrary, a condition of the pupils, precisely the reverse, is sometimes observed.

c. *Impairment of motion* is of more value in diagnosis, particularly the mode of its appearance and extent. The conditions, however, vary greatly. In one series of cases, actual paralysis did not occur at any time before death; instead, simple rigidity of the muscles was observed, or some twitching on one or both sides, followed later by muscular contractures. The hæmatoma in every such case was found on the opposite side. Small and large hæmatomata alike cause these symptoms; and here again it is owing to the different degrees of atrophy of the brain that in some cases the extravasation will destroy the function of the whole hemisphere, while in others it merely produces symptoms of irritation. In a second class of cases, paresis of one and then of the other extremity, and finally paralyses (hemiplegia), involving also the facial and hypoglossus, follow the initial twitchings, which are often quite slight. Frequently, however, the paralysis of the facial is the first paretic symptom, followed by the paralyses of the extremities. Many cases show contractures in a limited series of muscles, *e. g.*, those of the upper arm, after an initial paralysis or paresis. Often the opposite side remains unaffected until the entire cessation of the functions of the brain occurs. In a third class of cases the paralysis extends also to the other side—a fact of considerable value in diagnosis; for it is then evident that the affection is one which has spread to the other hemisphere, or that the extravasations have occurred at different times on the two sides. Just before the appearance of the paralysis on the second side, convulsive movements may first occur in the muscles of this side. Frequently the paralysis is only partial; it is rare that all the extremities and both facials are paralyzed before the total extinction of consciousness; this, however, happened in a case observed by us. Impairment of co-ordinate movements often occurs: as, for instance, uncertainty of gait, difficulty in writing and speaking, especially the latter (in nearly one-third of the cases quoted). In regard to this point, we are unable to say very much from personal observation. Hæmatomata are without doubt capable of causing difficulty of speech; true aphasia, however, we have never seen.

Many of the forms of impairment of speech, often attributed to the hæmatoma, are certainly due, not to the latter, but to the dementia paralytica.

d. Disturbances of sensation are not very common symptoms. Some cases, when hemiplegia becomes developed, exhibit dysæsthesiæ throughout the affected side, *e. g.*, formication, and numbness. But we have never met nor seen reported any case of impairment of sensation unaccompanied by loss of motion. In acute cases which lead to death a careful examination is manifestly impossible, owing to the loss of consciousness. Dysæsthesiæ of the extremities during the intervals between attacks are mentioned, but anæsthesia is not.

e. Fever occurred in all the acute attacks observed by us, except in paralytics with advanced atrophy of the brain. A marked type was wanting, but exacerbations and remissions occurred. Shortly before death there was usually, but not invariably, a decided exacerbation of the fever. The temperature is useful as an aid in diagnosis, simply in so far as it indicates the presence of fever, but not by reason of any type of fever which it may reveal. There is no fever during the intervals.

f. As the number of accurate observations regarding the *variations of the pulse* in this affection is very small, it is impossible to describe them even in the most general manner. Slowness of the pulse during the hemorrhage occurs in a majority of cases, and it is sometimes very marked. This symptom is, however, not constant. The pulse is often irregular, becoming more frequent near death. The rule, of course, is modified by the acuteness of the hemorrhage and the amount of compression.

g. The pupils.—The importance of contraction of the pupils during the acute attack has already been mentioned, as well as the coincidence of this symptom with drowsiness and severe headache. But this contraction, conjoined with feeble reaction or even complete immobility, is but transitory; after a continuance of the hemorrhage we have always noticed dilatation. Attention does not seem to have been paid to this point in many of the reported cases; the dilatation is spoken of by many as being unequal in the two eyes, the pupil opposite the side of the more copious extravasation being the more dilated of the two. Complete dilatation, as described by other observers (Durand-Fardel), we have never met. The initial contraction and immobility are to be regarded as symptoms of irritation ; the entire extinction of the functional activity of the centres of innervation follows so quickly, when the hemorrhage is a copious one, that a subsequent undue action of the sympathetic does not ensue. The pupil is therefore, shortly before death, entirely left to its own elasticity. It cannot be denied, however, that in some cases nothing abnormal occurs in the pupils.

h. Muscles of the eye.—We have never observed strabismus or ptosis. The symptom is mentioned by a few writers, but only in a very vague manner. We feel compelled to coincide with the opinion of Griesinger, that strabismus, and ptosis with extreme dilatation of the pupils, occur usually in ventricular and basilar affections ; while contraction of the pupils points to localized affections of the surface (commencing meningitis, superficial encephalitis, dementia paralytica, where strabismus is extremely rare). On the other hand, clonic and tonic movements of the eyeball upwards, and towards the side, do however occur.

The conclusions to be formed from the above facts are, that a dignosis is possible if the symptoms are well developed, and if the progress of the case can be shown to have been the same as that described above. There are, however, many non-typical cases ; for example, the patient may recover partially from the first attack, and then enter upon a chronic stage, gradually developing new symptoms, without any marked acute final stage. The intelligence may fail gradually from the first, and a progressive impairment of memory and judgment occur ; drowsiness is almost always present. Hemiparesis, numbness and difficulty of speech are slowly developed. The case may resemble exactly dementia paralytica, so that a diagnosis is impossible. We have in mind here those unusual cases of dementia paralytica—with which every alienist is familiar—where the characteristic psychical disturbances (excitement, hallucinations of grandeur) are entirely absent. In a case of supposed paralytic dementia, we found a double membranous hæmatoma without any large terminal hemorrhage, but with a number of small extravasations produced at different times, all in different stages of transformation.

VI. If some of the cases, mentioned in the last category, are, from the regularity of their course, capable of being diagnosticated, others occur which it is impossible to recognize. It is true that these cases are rare, at least when the disease is fully developed; they are usually not well defined, from the insignificance of the symptoms, which merely consist of slight attacks of loss of consciousness with fever, manifested during the course of a chronic affection of the brain.

7. Drinker, aged sixty, with atheroma and aortic insufficiency. July, 1865.—Violent headache; fever; excitement, soon giving place to drowsiness; unconsciousness; contracted pupils; nothing abnormal in the facial or hypoglossus; no strabismus; comatose during two days; recovers slowly, with weakness of left arm; a gradual diminution of intellect followed; memory poor; there was chronic headache, giddiness.

October, 1865.—A second attack, resembling the first, but of briefer duration; right facial paresis and paresis of right extremities; the latter, however, disappeared soon, leaving only the facial paresis.

After this, patient resumed his drinking habits; in the meantime he appears to have had a number of attacks; there is great drowsiness; paresis of right side gradually returned.

January, 1866.—Another attack of unconsciousness; fever; slow pulse; stertor; right facial and hypoglossus paretic; increase of the paresis of right side. Pupils contracted; that of the right eye somewhat the larger; twitching in all the extremities; coma; consciousness returned to a certain degree after forty-eight hours, but soon again disappeared; attack of croupous pneumonia; death.

Autopsy.—Large double hæmatoma, thicker on the right side than on the left; on both sides the organization consists of five or six membranes, loosely connected with the arachnoid and the dura, the latter being somewhat thickened; both hæmatomata show extravasations of different date, in the different layers—the largest extravasation being the latest and the one which had caused death; this was larger on the left side than on the right; atrophy of the brain; atheroma of the cerebral arteries.

A detailed consideration of the symptoms in this class of cases is unnecessary, as it would involve a mere repetition of what has already been stated.

Those points which have an important bearing upon the diagnosis and symptomatology of this affection, are the following:

I.—*The characteristic course.*

An acute diffuse affection of the brain occurs, which, notwithstanding the great severity of the symptoms, may be followed by a fair recovery ; an interval of good health follows, limited by a second acute attack, which may lead to death or to a second interval. A third acute attack may then terminate life.

II.—*The symptoms of the acute attack.*

a. Evidences of a sudden and increasing compression (headache, drowsiness, loss of consciousness, fever, characteristic pulse, sometimes violent initial symptoms of irritation).

b. Symptoms which indicate that the convexity is the seat of the lesion (contracted pupil, no impairment of the oculo-motorius and abducens ; no strabismus ; no ptosis).

c. Evidence of the spreading of the affection from one side to another (local symptoms on one side and then similar ones on the other).

d. Symptoms of compression of the whole brain (coma, disturbances of respiration, characteristic peculiarities of the pulse, extinction of the functional activity of certain centres of innervation, such as that which governs the act of swallowing, and that governing the motions of the pupil, and consequently the cessation of reflex movements of the pupil).

e. Symptoms of irritation of the motor centres of the surface of the brain, followed by paralysis of these centres (convulsions of one side, then of the other side, afterwards paresis of one side with paresis of the facial or hypoglossus of the same side, then paresis of the other side).

III.—*Symptoms during the interval.*

Symptoms of a process latent at times, causing irritation, however, and exerting compression, the effects of which vary greatly according to the intensity and the nature of the original disease (cephalalgia, diminution of intelligence, impairment of memory, drowsiness, partial paralyses, disturbances of speech, sudden mental excitement without cause, frequently mixed with symptoms of dementia paralytica).

IV.—*Predisposing influences.*

Usually old age, besides evidence of atrophy of the brain, resulting from alcoholism, atheroma, affections of the lungs, heart, and kidneys, chronic psychoses, or affections of the blood (anæmia perniciosa, hæmophilia, scorbutus).

Course.

It is difficult to give precise data as to the duration of pachymeningitis. No single individual possesses an adequate number of observations, and in the mass of recorded cases there is confusion between newly-formed membranes of old date and extravasations of blood, so that frequently the brief final stage is given as the whole duration of the disease. Of all the cases which have come to our knowledge, and of which the duration was recorded as carefully as possible,

34 per cent. had a duration of from 1 to 5 days.
40 " " " " 5 " 30 "
18 " " " " 1 " 6 months.
4 " " " " 6 mos. to 1 year.
4 " " " " over 1 year.

Few particulars in the published cases are given as to the duration of the various stages, and therefore any statistics that we might now collect would be of no value.

Terminations.

Pachymeningitis does not necessarily result in death. Although the majority of the cases have ended fatally, either from the extensiveness of the extravasation or from the original disease, yet there are a number of cases which prove that a comparatively large extravasation may be absorbed, that the new membrane may shrink gradually more and more, and that recovery may finally take place—only slight traces of the trouble remaining. This termination is uncommon; but among the brain affections, of which no diagnosis is ever made, are probably concealed not a few cases of this kind. Bouillon-Lagrange's case (quoted by Hasse) is evidence of this; Griesinger, Biermer, Textor, have met such cases, though they are not so convincing as that of Bouillon-Lagrange. Griesinger's patient[1] is still alive and in comparatively good health—with the exception of occasional headache and frequent sleeplessness, which may be regarded as recovery in comparison with the previous acute symptoms. The only objective evidences still remaining are, a slight left facial paresis and a moderate degree of feeble-mindedness. Such a result only occurs when the complicating morbid conditions, which lie at the foundation of the pachymeningitis, are of a transitory nature; or, in other words, if the brain, before the appearance of the pachymeningitis, has not already undergone too great a change (dementia paralytica, encephalomalacia, atrophia senilis, etc.); or, finally, if both hemispheres were not immediately compressed by the hemorrhage. There is no doubt that small extravasations, which occur in other diseases, are frequently absorbed. This is proved by the delicate new membranes (sometimes colorless, sometimes colored with hæmatoidin crystals) which are frequently found on the inner surface of the dura, in all the affections mentioned above. In support of the view that large extravasations of blood may be absorbed, we would point to the more voluminous deposits of a similar nature, which are occasionally seen. These membranes, which are composed of many layers of connective tissue, easily separable from each other, are separated from the arachnoid by newly formed epithelium, but in a few places delicate adhesions bind them together. The

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[1] Archiv der Heilkunde. 1862.

Prognosis

is, therefore, not absolutely fatal; recoveries, however, are quite rare. The unfavorableness of the prognosis, however, is mainly due to the causative affection, as is evident by the number of shrunken pachymeningitic patches which have been observed; death is in these cases caused by the original disease.

Treatment.

Those who have had the opportunity of trying the effect of treatment in diseases of the brain, will not have great expectations of success in dealing with hæmatoma.

Where there are grounds for suspecting the existence of this disease, general symptomatic treatment should be employed. Derivatives, abstraction of blood, cold, stimulants, narcotics, cathartics, are the means usually employed.

Large extravasations are as little subject to treatment as are large apoplectic hemorrhages into the substance of the brain. An energetic antiphlogistic treatment may be adopted, and the whole category of derivatives may be employed, but no satisfactory results will be obtained. The hemispheres bear a certain amount of pressure, which becomes equalized in some unknown way. This does not occur, however, if the extravasation is immoderately large, and, under such circumstances, any efforts of our own to relieve the difficulty would, as a matter of course, prove futile.

Different indications present themselves at the various stages of the processes, which have been mentioned before as occurring in the course of a case of long duration. It is particularly desirable to determine the primary evil (disease of lungs, heart, kidneys, blood), and employ the treatment usual in these affections.

Treatment of chronic alcoholism will often be demanded. It need hardly be mentioned that the treatment of senile involution of the brain, dementia paralytica, and other forms of atrophy of the brain, is likely to be very unsatisfactory.

The affection itself, during the hemorrhage, should be treated antiphlogistically. It cannot be expected that the abstraction of blood will cut short the attack, but every means should be employed to diminish compression.

Therefore, leeches are to be put upon the temples and behind the ears, cups on the neck. The abstraction of blood should be ample, in order to be of any benefit; the strength of the patient must, however, be considered. Venesection even is not to be rejected in strong patients. We have seen cases where its employment, in a series of attacks, was the only thing that afforded any relief. The best plan is to perform but a single though copious venesection; after it, the coma appears to be less profound, and compensation of the pressure of the extravasation seems to occur more readily, unless, of course, the amount of the extravasation is too great. The energetic use of cold, the application of the ice-bag, is never to be omitted—an application agreeable to patients after they recover from the coma. Some derivative should at the same time be used. We are inclined to disbelieve in the use of irritants on the skin of the head or immediate vicinity; they can, however, be employed as a temporary measure on the extremities, or on the trunk; for this purpose sinapisms, irritant baths, applied to the hands and feet, Junod's boot, which we used on one occasion, seem to have been of service.

If there are no contra-indications, derivation to the mucous surface of the intestinal tract is to be recommended: clysters, large active doses of calomel and senna, or saline laxatives, and castor-oil. During the acute attack, the patient should be put under the most favorable conditions; all excitement should be avoided; the use of further internal medication is to be sparing, and the fact held in mind that we are unable to check the hemorrhages, though the symptoms caused by it can without doubt be modified.

It might be imagined that medication would be of benefit during the period following a recovery from the attack. Experience teaches the reverse. The regulation of the habits of life, of diet (*e.g.*, in drinkers), and the removal of all causes influencing injuriously the primary affection, are to be carefully attended to. Strong drinks, too high living, mental or physical exertion, are to be avoided. All so-called absorbents have proved ineffectual in our hands. Intercurrent cephalic attacks are to be treated antiphlogistically, the impairment of strength to be combated; sleeplessness is to be treated by the rational use of narcotics; and the vis medicatrix naturæ, in effecting a resorption of the membrane, is not to be forgotten.

II. Affections of the Pia Mater.

Arachnitis has been described in all text-books until within a short time. It will not appear strange, however, if it be omitted here.

Pathological changes in the so-called visceral arachnoideal layer (we have already stated our opinion regarding the parietal layer) are not rare. But this visceral layer (external thickened layer of the pia) is not subject to disease by itself; it serves simply as a bridge for the transmission of morbid processes from the bone and dura to the deeper tissues. A direct transmission from membrane to membrane (which in life are probably not in contact) is rare. The tubular sheaths through which the nerves pass are much more frequently the means of communicating the disease (meningeal affections in caries of the petrous portion, and suppurative pachymeningitis).

Even the older authors were sceptical as to the existence of arachnitis as a separate inflammation of the external surface of the "visceral arachnoideal layer;" later writers (Rokitansky, Förster, Rindfleisch) deny it entirely. In fact, no such independent affection exists. It is true, however, that changes are occasionally seen in the external surface of the pia (visceral arachnoideal layer) in inflammation of the pia. But then, when intense inflammatory changes are going on in the pia, when the whole tissue is permeated with pus cells, why should not some of them find their way to the external surface of the thickened limiting membrane? We have frequently, under these circumstances, observed on the external surface of this membrane, a thin layer of true pus, which could be scraped off with the knife. Many similar observations have been made by others.

Some writers mention that they have found a turbid fluid between the dura and pia (in the so-called sac of the arachnoid). This is interesting, as Hitzig has shown that during life a certain quantity of cerebro-spinal fluid remains between the dura and pia, which disappears quickly after death. Under certain circumstances, however, as it appears, this disappearance of the fluid does not take place.

Certain chronic changes may occur in this locality, but they have no clinical value: thickening, hypertrophy of the connective-tissue basis substance (dementia paralytica, chronic alcoholism), chalk deposits of small extent, deposits of pigment, etc.

A.—Hyperæmia of the Pia.

It is impossible to separate passive congestion and fluxion of the pia from analogous conditions of the adjacent brain. All attempts to make such a distinction have proved unsuccessful. The cortex and the larger part of the white substance of the hemispheres are so dependent upon the vessels of the pia for nutrition, and the connection is so close, that congestion in the latter, not dependent upon local causes, is associated with congestion of at least the upper half of the corona radiata—usually in a less degree, from the greater resistance the adjacent substance of the brain offers. The conditions for hyperæmia are therefore more favorable in the pia ; indeed, after the displacement of the cerebro-spinal fluid, the congestion may attain a high degree. In the brain the variations are more limited ; the vessels can be dilated until their walls come in contact with those of the perivascular space, but no further, since we believe the brain of the adult is incompressible. In certain diseases (senile atrophy, for example) the perivascular spaces may become wider, and a greater dilatation of the vessels is thus made possible. This is a fact, however, difficult of anatomical demonstration, though it undoubtedly may occur in high grades of passive hyperæmia.

It is irrational to regard all disturbances of the circulation from one standpoint. The fluxionary hyperæmia of childhood cannot be considered identical with apparently similar changes in old age, except by those who do not aim at clearness in the pathology of these affections. An attempt will here be made to consider the different causes before forming a classification. The uncertainty in this matter depends upon the following facts :

1. In the cortical portion of the brain, it is simply impossible to distinguish an arterial from a venous hyperæmia. The general dilatation of the cortical vessels and the contraction of the perivascular spaces can be demonstrated by subjecting the parts to certain preparatory processes ; but we do not think any one has been able to separate in the cortex hyperæmia of the arteries from that of the veins—especially in those arteries and veins which pursue in the main a vertical course.

2. The same is true of the upper layer of the corona radiata. The color is no help, as this is quite deceptive in the cortex. The white substance becomes rosy-red in color, if there is an injection of the small arteries, and bluish-red if the small veins are injected. The nearer one approaches to the groups of ganglia (and in the same), the easier it is to distinguish between the two kinds of congestion. Here the larger vessels are all veins; and arteries of large size, as it seems, are not to be found in the substance of the brain above the level of the anterior commissure.

3. In the pia the difficulties are not quite so great, as the arteries can be traced from the base of the brain. But another obstacle is encountered here. It has for a long time been believed that hyperæmic arteries discharge, at the moment of death, their contents into the veins, the action of the heart suddenly ceasing, and no further pulse wave being driven forward. If, after the heart's action has ceased, the arterial vessels contract again, no estimate as to whether they are congested or not is possible. Physiological proof of this has been given by Goltz, Thiry, and Bezold. This, however, cannot always be the case, otherwise an arterial hyperæmia would never have been observed.

4. The difficulty is increased by the peculiar relation which hyperæmia of these parts bears to œdema of the neighboring tissues. As soon as the capillaries are dilated, either from the venous or from the arterial circulation, a transudation occurs. This, in the cortex, causes œdema—in the meshes of the pia, a collection of fluid; the latter also takes place in the ventricles. Great caution, however, should be used in utilizing these morbid appearances as a means of explaining the clinical symptoms. Certain forms of œdema undoubtedly arise only at the moment of death; and this is especially true of œdema of the brain. Chronic œdema may occur in old people, simply compensating for the shrinkage in the brain.

5. Hyperæmia of the pia and cortex frequently disappears when pressure is subsequently applied. The best example of this is to be found in the anæmia of the pia and cortex in tuberculous meningitis, when a copious exudation into the ventricles crowds the cortical portion up against the inner surface of the

skull. A similar condition follows in the fluxionary hyperæmiæ of childhood, which lead to a simple ventricular effusion; the initial hyperæmia is so completely masked, that its existence has only been demonstrated in a few autopsies of children who died during an early stage.

6. The recognition of many cases of fluxionary hyperæmia is impossible, as the heart near the end of life loses its power, and for hours contracts only imperfectly, causing a condition exactly the reverse of the original one; that is, an arterial anæmia and venous hyperæmia ensue, not at all in accordance with the symptoms during life.

Before touching upon the pathological anatomy of this condition, we shall discuss the causative influences, as it is only in this way that the different conditions can be well classified.

It has always been customary to divide hyperæmiæ of the brain and pia into fluxionary and obstructive (active and passive, primary and secondary) hyperæmiæ. In old age, as will hereafter be seen, this distinction is at times valueless, but in general it is to be adhered to.

a. Passive hyperæmia. In a large number of cases organic diseases of the heart and lungs are the causes of this state. Diseases of the lungs, which, by compression or by structural changes, impede either temporarily or permanently the pulmonary circulation, lead in all ages to venous congestion of the brain. If at the same time the aspiration of blood be lessened by obstruction to inspiration (rigidity of the thorax, emphysema, deformity of thorax), the cerebral congestion is greatly increased. It is not our province to describe the different mechanisms which characterize the different affections of the lungs and pleura; we shall simply mention the mechanisms which provide a certain degree of compensation (hypertrophy of the right and left heart, and the thyroid body acting as a compensating blood reservoir, according to Liebermeister and Guyon). Temporary relief may thus be afforded, but in incurable affections it is insufficient.

All the processes which cause laryngeal stenosis also lead to hyperæmia of the brain and pia. If the inspiration alone is obstructed, a great degree of venous congestion can hardly occur, unless the blood have already become overloaded with carbonic acid. If from any cause expiratory dyspnœa set in, the veins of the neck and head become distended with blood. The thyroid body may here also exert a compensating influence. Affections which cause impediments to the pulmonary circulation, as diphtheritic croup, increase this venous stasis. As soon as the amount of carbonic acid in the blood passes a certain limit, weakness of the action of the heart sets in, still further increasing the difficulty. A similar state may, of course, be produced by other affections than croup and diphtheritis.

The most marked instances of venous congestion result from affections of the heart. Without entering into a very accurate enumeration, we will simply mention: the true venous pulse in insufficiency of the tricuspid, the decided changes in the circulation caused by mitral stenosis, the dilatation of the small veins in various forms of congenital pulmonary stenosis, the final stage of all valvular lesions, and the different parenchymatous degenerations of the heart. Temporary compensation sometimes occurs, as is well known. Naturally, congestive hyperæmia of the pia, dependent on acquired diseases of the heart, is more frequent in old age; while that connected with affections of the lungs may occur at all ages.

In childhood a number of special conditions lead to analogous changes in the pia. Acute infantile diarrhœa is frequently attended by venous hyperæmia of the pia and brain; at least, this is the condition found in a number of those cases where death occurs with hydrocephaloid symptoms. There is reason to believe, however, that arterial anæmia is the cause of death in these cases; the clinical symptoms should not, therefore, be referred to the congestion of the veins found after death. As in the majority of cases, so here also this congestion is without significance; it is simply the necessary consequence of enfeeblement of the heart, and is often not found in extremely emaciated children, where so great a degree of anæmia results from the chronic disease, that when they finally die from the diarrhœa there is not blood enough left for venous congestion. Marked venous congestion of the veins of the pia and of the sinuses is found, for the same reason, in infantile cholera and in cholera of adults.

In childhood, hypertrophy of the thymus, which occasionally occurs, is of importance, as are also large glandular tumors of the neck. Obstructions in the veins themselves, such as thrombosis of the superior vena cava, of the internal jugular, and of the sinuses of the brain, should also be mentioned. The latter sometimes hinder the discharge of blood to such a degree, that apoplexies may result.

Finally, passive congestion in the tract of the vena cava superior may occur at all ages, if any compression in the abdomen (from ascites, peritonitis, tumors) exists.

Venous congestion of the pia is frequently found in children dying of acute febrile affections, without such brain symptoms having manifested themselves as would imply the presence of certain anatomical appearances. High fever, the resulting weakness of the heart, which sets in shortly before death, and the consecutive anæmia of the brain, give rise to a series of well-known nerve symptoms; but it is not to be inferred that these latter are due to the venous hyperæmia found on autopsy; this state necessarily results from the causes already mentioned—first, the weakness of the heart, and, second, the emptying of the arteries at death, on the cessation of the heart's action. It is an established fact (the reasons will be stated further on) that the amount of cerebral blood is subject to greater variations in children than in adults.

b. Fluxionary hyperæmia. If we consider the conditions under which an active congestion may occur, it will at once be evident that the different periods of life must be examined separately. We are satisfied that fluxionary hyperæmia of the pia and brain may occur in children under circumstances not influential in old age, or only so to a less degree. We must, therefore, seek for other causes for this condition in adults. Finally, certain forms of congestive hyperæmia occur in old age, which in their duration, their effects, and their mode of origin, cannot be compared with any of the forms which occur at other periods of life.

A. *Fluxionary hyperæmia in children.*—Two causative conditions are to be distinguished. An increase in the fullness of the blood-vessels occurs in a vascular tract if the action of the heart is temporarily increased, while the elasticity of the vessels remains the same. The resistance of the vessels will be overcome, they become dilated, and *hyperæmia* results. Again, the vessels may become dilated by reason of a diminution in the vigor of the vaso-motor nerves, the action of the heart remaining the same. As a well-known illustration of this we may point to the change produced by section of the sympathetic, and the consequent fluxionary hyperæmia of the part thus deprived of its vaso-motor influence.

The first factor (temporary increase of the power exerted by the heart) is much more important in childhood than in adult age. It is known that the vessels of the brain in infancy possess a great delicacy of structure and extensibility, almost resembling in this respect vessels which have undergone a pathological change (Virchow). Perhaps, too, the greater softness of the brain in childhood may allow a slight compression. This latter is, however, of less importance, from the fact that the sutures are not fixed, and the covering of the fontanelles in infants allows an increase in the size of the cranial cavity.

Under these conditions any temporary increase in the force of the heart's action will give rise to a transitory dilatation of the cerebral vessels. The duration, degree, and consequences of this (escape of the serum of the blood, migration of white corpuscles, rupture) will depend on the nature and duration of the abnormal state of the heart. The symptoms of fluxionary hyperæmia of the pia and brain coincide, therefore, very closely with those which are observed in a moderately severe access of fever. Symptoms of greater severity, such as result from the sudden rise of temperature, may also be explained by the dilatability of the cerebral vessels. If these conditions persist for a longer time, *true inflammatory changes* are developed. The second of the above-mentioned influences is also of importance in childhood. The small arterial vessels of the pia and the brain are said to contain no nerves; the capillaries indeed are not provided with nerves. It cannot, however, be supposed that no vasomotor influence is exerted upon the cerebral vessels. It would be of the greatest importance in pathology to know all the circumstances under which the cerebral arteries dilate or contract.

Known facts in regard to this point are, however, few; they are the following:

1. The vigor (tonus) of the vaso-motor nerves of an arterial tract is impaired by a strong irritation of the sensitive nerves of this portion (Lovén). Lovén's experiments showed that usually there is at first a contraction of the vessels, followed by dilatation; the latter state lasting a much longer time. (Experiments on the arteria auricularis.) In the majority of cases dilatation was limited to the tract supplied by the sensitive nerve irritated. Sometimes, however, this limit was exceeded. From a number of pathological facts there can be no doubt that this is particularly liable to occur in the head. We think also that many painful affections in the neighborhood of the cranium in this way cause transitory hyperæmia of the cerebral vessels, as hyperæmia of the pia following congestion of the conjunctiva, angina, or facial erysipelas, hyperæmia of the pia accompanying aching teeth in children, and painful affections of the mucous membrane of the mouth. These causes are all active in adults, but in children the cerebral vessels are more delicate and possess thinner walls; hence dilatation of the vessels of the pia occurs more easily in early life. A case which came under our notice illustrates this: a child, delirious from facial erysipelas, died suddenly from a sudden and very marked rise of body-temperature; the hyperæmia found after death in the pia was evidently arterial in character.

2. Changes in the calibre of the cerebral arteries occur on irritation of even a distant sensitive nerve. Nothnagel was the first to show that the irritation of a distant part of the skin caused contraction of the cerebral vessels by reflex action. The cervical sympathetic, the ganglion supremum, and the intracranial centres were found at the same time to be the routes through which the vaso-motor nervous supply was furnished to the arteries of the pia. The value of these observations has been questioned (Riegel and Jolly); but our own experiments confirm Nothnagel's statements. We would add, however, that the vascular contraction mentioned by him is quite transitory, and is followed by dilatation of longer duration; and, further, that the effect varies greatly in intensity and duration, according to the site and

intensity of the irritation, and that the same effect may be produced if an irritant is applied in the abdominal cavity (peritoneum and intestine). If this view be once generally accepted, an explanation will easily be found for the fluxionary hyperæmia of the brain and pia which occurs in many affections of childhood. This condition is common in digestive disturbances, catarrh of stomach and intestine (so-called from ignorance of the exact state). The nerve endings in the intestine are irritated in some way, probably by the decomposed ingesta and the intestinal fluids. Severe pain elsewhere in the periphery is also capable of producing a similar condition.

3. Perhaps a violent though transitory contraction of the arteries of the intestine and peritoneum may be influential in causing the fluxionary hyperæmia under consideration. Cold applied to the stomach irritates the vessels throughout a wide extent to contraction, and in this way the pressure in the cerebral vessels is greatly increased. Probably other irritants act in a similar way; but of this we have no positive proofs. The experiments of Schüller deserve mention in this connection. Cold applied to the abdomen caused immediate dilatation of the cerebral vessels. Heat applied to the same place caused contraction of these vessels. Cold and warmth (cold and warm baths) applied to the entire surface of the body have similar effects. Packing with moist cloths first causes dilatation and then gradual contraction. Cold applied to a spinal nerve, laid bare, caused contraction. The pressure in the carotids is increased by cold applied to the abdomen.

4. The sudden dilatation of vessels, sometimes preceded by a general transitory contraction (pallor from anger, and at the same time a violent action of the heart, afterwards sudden flushing), which occur from mental excitement, are also of importance in childhood. Eclamptic attacks are not infrequent after angry excitement, in children afflicted with great deformity of cranium or cerebral affection. The symptoms of cerebral hyperæmia are, it is true, in these cases, not so apparent as those of the initial but transitory contraction of the vessels.

B. *Fluxionary hyperæmia in middle age.*—The variations in the amount of blood in the cranium cannot be as great in adults as in children. The vessels are not so easily distended, the substance of the brain is not compressible, and the capacity of the cranium cannot be increased. Consequently, there remains only the possibility of displacement of the cerebro-spinal fluid. Still, true congestion of the brain does occur in adults:

1. In chlorosis, anæmia, retarded development. Rokitansky called attention to a peculiar weakness and delicacy of the vessels in chlorosis. Virchow also mentioned it, and added greatly to our knowledge of the subject by a careful description of the premature fatty degeneration of the vessels under these circumstances. Any accidental over-activity of the heart will have therefore the same effects as it has in children. Further, Virchow and others have pointed out a congenital narrowing of the aorta in chlorosis, which necessarily causes hypertrophy of the left heart. A moderate true hypertrophy of the left side of the heart is not uncommon in chlorosis, and in such cases (which are not confined to the female sex) these peculiarities constitute a fertile source of congestions of the pia and brain.

2. Intense and protracted congestion of the pia and brain very frequently occurs in hysterical persons. The changes are evidently the same as those previously mentioned, namely, a momentary contraction of the vessels, followed by long-continued dilatation (a transitory capillary pulse and dilatation of the vessels have been observed by us in the retina in these cases). They are apparently excited with the greatest ease and intensity, particularly by mental excitement, in persons suffering from hysteria. The convulsions of hysteria, excited by anger, fright, and the like, are well known, and it is also known that these attacks are followed by prolonged and intense cerebral congestion. Hysteria has been mentioned as an example, but in all psychopathic patients a similar predisposition exists.

3. In middle age hypertrophy of the left ventricle is a fertile source of congestion, from whatever cause the hypertrophy may arise.

Hypertrophy of the left ventricle, consequent on aortic insufficiency, is followed by a dilatation of the whole arterial system, from which, as is known, we gather a number of diagnostic signs; this dilatation indicates a constantly increasing loss of elasticity of the vascular walls, which in its turn tends to increase the hypertrophy. Hence arise arterial fluxions to all parts of the body, those to the head and neck being particularly distressing to the patient. The same is true of the compensatory hypertrophy accompanying cirrhosis of the kidney, which becomes a source of numerous dangers, especially when, in consequence of its complication with a widespread atheroma, apoplexy is threatened.

4. Without doubt, the excited action of the heart during fever is of importance during this period of life, but much less so than in childhood, since great variations in the quantity of contained blood are not possible in the adult cranial cavity. Suspicious symptoms accompanying high fever in adults can always be explained on the supposition of the influence of the poisoned blood upon the central organs. The only exception to this is the chill, during which the vessels of the skin are contracted, and congestion of all the inner organs, including the brain, necessarily occurs. The latter manifests itself by very striking symptoms.

5. Violent irritation of peripheral nerves. Sudden and violent pain is not infrequently observed to have a marked effect upon the functions of the brain.

EXAMPLES.—After a circumcision, done rapidly, without narcosis, the patient became suddenly pale, and lost consciousness; general convulsions of a few seconds' duration occurred; the face and conjunctiva became flushed; consciousness returned immediately; there were violent headache and all the symptoms of a severe transitory congestion.

At the moment of the perforation of an ulcus ventriculi with sudden and very severe pain, the patient fell down unconscious; brief general convulsions ensued, followed by return of consciousness, and for twenty minutes by symptoms of congestion, headache, and confusion of ideas. Peritoneal collapse then set in.

In these cases we necessarily think of a brief contraction of the cerebral vessels, followed by dilatation. It is not known whether this may result from irritation of all peripheral nerves. Individual disposition is perhaps to be considered as an influential factor.

6. Cerebral hyperæmia frequently accompanies painful affections in the neighborhood of the cranium, ex. gr. erysipelas.

Some, however, consider it to be then simply a collateral fluxion, resulting from the disturbance of the circulation in the affected parts.

7. The depressor vagi (Cyon, Ludwig) seems to have no direct effect on congestion. Indirectly, the whole arterial system can be dilated through the influence of this nerve on the action of the heart. The heart's task again is diminished after dilatation of the peripheral vessels. The influence of this interesting correlation on the pathology of this affection is as yet but little known.

8. Congenital stenosis of the aorta, at the point of insertion of the ductus Botalli, is regarded by Hasse as one cause of frequent intense fluxions to the head. The conditions, under these circumstances, are exceedingly favorable to arterial hyperæmia, but not equally so in all cases, as would appear from the absence of hyperæmia in a case coming under our observation.

9. Poisoning. After the absorption of toxic substances, changes occur similar to those already described. Venous hyperæmia of the pia and brain is one of the most frequent. This is not, however, due to the direct local action of the poison, but results from paralysis of the heart.

The congestion of the pia and brain depends here also upon paralysis of the vaso-motor nerves supplying the cerebral vessels, upon increased action of the heart, and, finally, upon collateral fluxion, consequent on contraction in large peripheral arterial tracts. Here, too, the arterial hyperæmia is very difficult to demonstrate after death, for reasons that have been already given. There can be no doubt, however, that true arterial hyperæmia occurs during life, for other arterial districts in the head which are exposed to direct observation have been seen to be in a hyperæmic condition, and moreover cerebral apoplexies have been found in cases of accidental and experimental poisoning.

The difficulty of reconciling these anatomical facts with the clinical symptoms is even greater here than under other circumstances, for the reason that most poisons exert some specific action as yet unknown upon the nerve substance itself.

The predicament is therefore the same as in the acute febrile diseases; it is difficult to decide whether the brain symptoms are due to fluxionary hyperæmia or to the vitiated state of the blood.

10. *Sunstroke.*—This furnishes a striking example of the difficulty of determining the state of the arterial vessels. There can be no doubt that at the onset of the symptoms there is great hyperæmia of the pia and brain, but no trace of this can be found after death; on the contrary, extreme anæmia of the pia and brain (Arndt) is observed, with œdematous infiltration of the latter, and great distention of the whole venous system. Other organs present similar appearances. Many authors have, without doubt, mistaken venous for arterial injection; but the capillaries are almost entirely empty, and the blood is crowded into the veins. Recollecting that experiments have shown that moderate heat, directed upon the cranium, causes dilatation of the vessels, we must conclude that the initial hyperæmia of sunstroke is due to heat. This, however, is but the least of the changes produced by insolation. Enough facts are not yet established to justify any decided opinion as to the pathology of this affection. Accurate anatomical investigations in the various stages of the affection are required. Arndt's observations are sufficient to show that a fluxionary hyperæmia can entirely disappear under the pressure of the fluid transuding into the tissues; the character of the cardiac contractions (frequent and weak, at times irregular) must also be regarded as aiding to bring on this condition.[1]

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[1] Arndt believes that a parenchymatous degeneration (cloudy swelling) of the liver, kidney, and heart is rapidly developed under the influence of the enormously high temperature in this disease. [This is, however, not yet proven.] He generalizes from this to the brain, and speaks of a diffuse encephalitis, not yet, however, demonstrated. In this way he explains the cerebral symptoms which often remain long after the acute attack. The blood is acid during an attack of insolation, is rich in urea, is very rich in white globules, and shows very little tendency to coagulation. Probably lactic acid causes the acid reaction. Köster's observations also show that the pathology of sunstroke is still open for investigation. He found affections of the superior sympathetic ganglion and of the vagus; swelling, hemorrhages, separation and destruction of the nerve fibres, extravasations in both vagi and both phrenic nerves. He also found a cerebral hyperæmia, but does not give particulars as to its quality and results.

11. Vaso-motor paralysis of the cerebral vessels (Morbus Basedowii).

This condition of the vessels leads to great distention of the cerebral arteries; fortunately the arteries of the thyroid are also involved, as otherwise the effects on the brain would be much more serious. Psychical irritation is the direct result of it. Some are inclined to explain by this vascular distention the exophthalmus and dilatation of the pupil, assuming a direct congestive irritation of the centre for dilatation of the pupils (nerves to the muscle of Müller, to the dilatator pupillæ).

C. *Fluxionary hyperæmia in old age.*—Degeneration of the arteries, including atheroma and ulceration of the intima of the larger and middle-sized vessels (circulus Willisii), calcification of the media, and, finally, degeneration of the small cerebral vessels, even to the calcification of the capillaries, which sometimes affects all the capillaries of the cortex and the corona radiata, is added in old age to the predisposing influences already mentioned. The hyperæmic state is then often chronic, and slowness of the pulse frequently accompanies the change in the arteries—a condition of things very different from what exists in the ordinary forms of fluxion.

The brain in old age is atrophic. Its nervous elements undergo a destruction, many traces of which, in the shape of degenerated tissue elements, are found. The vessels do not take part in this destruction, and for that reason Durand-Fardel thought that the senile brain contained more vessels than the normal brain. This atrophy may depend upon the vascular degeneration (a fact difficult of demonstration), or it may be the result of some unknown influences; in any case, however, a void will be created in the cranial cavity. This void will, it is true, be filled by some compensating fluid, but still greater variations in the sizes of the vessels will be possible than during middle age. There exists, therefore, a certain analogy with the period of childhood, though the differences both in nature and in results are great.

In general, throughout the whole circulatory system, the intima is thickened and rough; there is a loss of elasticity, and a permanent dilatation of the vessels is in this way caused. In consequence of this loss of elasticity, an increased amount of work is thrown upon the heart, as the non-elastic arteries are not able to aid in propelling the blood. The left ventricle becomes consequently hypertrophied. In the following corollaries are contained all the conditions that favor the production of arterial hyperæmia of the brain in old age :

a. If the atheroma extend, as it frequently does, to the bifurcation of the carotid, or to the branches of the circle of Willis, the resulting loss of elasticity of the arterial walls causes a loss of a portion of the propelling power of the heart, and the cerebral circulation will be retarded in consequence.

b. The same effect on the circulation in the small vessels of the pia and brain is produced, when the vessels in question are narrowed by atheromatous degeneration. But this is merely temporary, for the rapidly developing hypertrophy of the heart causes dilatation of the inelastic vessels, and compensates for their loss of propelling power. The smaller vessels may then regain almost completely their normal conditions.

c. As long as the muscular substance of the hypertrophied heart is normal, the patient is liable to congestion from increased action of the heart. But fatty degeneration soon sets in and impairs the force of the heart's action ; the circulation in the cerebral vessels becomes slower ; marked venous with but slight arterial congestion is gradually established, if the heart be much changed.

d. The above remarks are also applicable when the degeneration extends to the smaller cerebral arteries. Even when the retardation of the circulation in them is temporarily counterbalanced by the cardiac hypertrophy, they do not escape dilatation. As long as the heart retains its propulsive power, all the conditions of chronic hyperæmia are present. This state is indicated by the formation of aneurisms on the small arteries, by the great number of bloody points, corresponding to transversely divided vessels, found on section of the brain, and by the dilatation of the perivascular spaces, so easily seen in the region of the ganglia, even when the hyperæmia has disappeared at the time of death.

e. When the degeneration affects only the small arteries of the brain, the changes are the same. As soon as the small vessels become dilated, the conditions necessary for the establishment of chronic hyperæmia exist.

f. If only a part of the cerebral arteries are atheromatous (the one most frequently affected is the arteria fossæ Sylvii), the initial narrowing will necessarily cause a fluxion to the other branches of the circle of Willis. The gradual development of the atheromatous change permits the establishment of a sufficient circulation through the anastamoses of the vessels of the pia, and no symptoms are to be expected. As soon, however, as the diseased arteries become dilated, all the conditions necessary for chronic hyperæmia of them are furnished.

As, however, in this form of hyperæmia, slowness of the blood-stream plays an important part, and as thereby a new and very important etiological influence is added, it is evident that there is a great difference between it and the previously described variations in the calibre of the cerebral arteries. Its consequences are also very different; but a consideration of these (apoplexy and softening from idiopathic arterial thrombosis, embolism by particles of atheroma, partial cerebral œdema, œdema of the pia, chronic hydrocephalus ex vacuo) does not come within the limit of this article.

The *pathological anatomy* requires but a brief consideration. Venous hyperæmia of the pia and brain is easily recognized. The sinuses of the dura are distended with blood, as are also the veins emptying into the sinuses: if the congestion has lasted any length of time, the veins are dilated, tortuous, and present varicosities here and there. Degeneration of the walls has been frequently observed, and occasionally, if the congestion has persisted a long time, a marantic thrombosis is produced. The capillaries are distended, as has been proved by measurements. We have in a few cases found varicose enlargements of the capillaries of the cortex and the corona radiata, filled with a collection of white corpuscles, such as are seen in leukæmia, or in the retina in pernicious anæmia. They were not produced simply by increase of blood pressure; more complicated conditions were also present.

Under these circumstances a dropsical condition of the brain and the pia is developed; but we think that a certain amount of atrophy is requisite for it. At least we have satisfied ourselves, after a number of autopsies on young patients with lesions of the heart, that no considerable collection of fluid can occur in the cranium when the brain is healthy. We believe, however, that the continuous venous stasis may be the exciting cause of cerebral atrophy; still this view requires special confirmation, because the impeded nutrition and ultimate marasmus, following diseases of the heart, affords a sufficient explanation of atrophy of all the tissues. At any rate, in old people, when atrophy of the brain exists, from whatever cause, œdema of the pia and brain and hydrocephalus internus are never wanting, if congestion has been present. It is not easy to ascertain the cause of the marked thickening of the pia, of the firmness and thickening of the ependyma of the ventricles, changes which almost all chronic stases ultimately bring about. Atrophy of the brain coincides with these too, almost without exception. The simple explanation, that the serum transudes in venous stasis, and that a species of "hypertrophy ex vacuo" results, does not seem satisfactory. In the pia, the change is not merely an increase in volume from the dilatation of the vessels, or from œdema of the tissues, but there is a real increase of the tissue-elements themselves.[1]

That a hyperplastic process takes place cannot be denied, and in this we have an analogy with the processes in other localities where great venous stases occur, and are, as is well known, frequently accompanied by active hyperplasia.

Arterial hyperæmia, as may be inferred from what has been already said, has very rarely been observed on the cadaver. It is most easily studied on the pia, where the large arteries can be followed for a short distance. If this can unquestionably be done it is fair to conclude that we have not a transitory fluxion to deal with, as that results from temporary causes, which cease at death. It is especially in chronic processes, as in Basedow's disease, that the chronic arterial hyperæmia comes to view. But, in fact, the transudation of fluid from the stagnating blood column, which begins as the heart's action fails and finally ceases, almost completely obliterates the pathological changes. The mass of the brain seems larger and swollen; the sulci are effaced, and the convolutions flattened. This may be very noticeable. When the serum of the blood has transuded, and at the same time the pressure in the brain substance has, in consequence of the cessation of the heart's action, become much greater than that in the arteries, the recognition of the hyperæmia becomes impossible. The vessels collapse, and a pia, which was very hyperæmic, intra vitam, may appear quite anæmic after death. This causes a great deal of confusion in the pathology of cerebral œdema also. We are inclined to believe that it is impossible to distinguish the cerebral œdema occurring at the time of death from that which has occurred during life, except where evident inflammatory changes are present.

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[1] We have observed specimens of simple venous stasis in which the occurrence of migration could scarcely be denied. This can easily be explained, if the slowness of the circulation, amounting, in places, to complete stasis, be kept in mind.

The conditions of pressure, both in the vessels and in the brain, that come into question here will be discussed later.

Frequent and long-continued attacks of chronic arterial hyperæmia of the pia cause hyperplasia, as has been already mentioned. The changes here are even more easily overlooked. But we are now touching on the border-line between simple hyperplasia and true inflammation. This matter will be more fully discussed in the following chapters.

Symptomatology.

It is extremely difficult to differentiate in a rational manner the symptoms of the different varieties of hyperæmia of the brain. A number of different forms are described by many (Andral, Durand-Fardel), which we cannot consider as really instances of simple fluxions. In the causation of some sets of symptoms (particularly convulsions in adults) it is evident that other influences are at work besides the congestive hyperæmia. When we glance over the various symptoms that can be attributed to simple congestive hyperæmia, we find that they all have something in common, viz., they are transitory and leave no permanent effects. Otherwise, however, they differ so greatly, that it is manifest that the special conditions under which they are produced cause the various classes of cases to assume very different forms. In the first place, persons of all ages and all conditions of life and health are attacked, children and old people, persons with fever, and persons without. During middle age, too, the causes of the hyperæmia are so exceedingly various (hysteria, aortic insufficiency, cirrhosis of the kidney, poisoning), that it would seem impossible to construct a clinical history that would be characteristic of all cases. The difference in the nervous sensitiveness of various temperaments is to be also remembered. Psychopathic women may present the symptoms of an inflammatory cerebral affection, or an acute psychosis, during a slight febrile increase in the heart's action; the same process may in one person cause drowsiness, or unquiet sleep with wild dreams, and in another sleeplessness and great psychical restlessness. Healthy people will suffer from severe headache during a sudden congestion of the central organs; but paralytics, with atrophy of the brain, are not disturbed by it. The effects of congestion in adults are not the same as in children. Finally, everything depends upon the quickness and the violence of the process. Extreme congestion seems to be able to put a temporary stop to the functions of the brain, but we cannot as yet say that some of this effect is not due to a slight œdema of the brain. The pathology of a coup de sang is likely to remain for a long time a puzzle.

a. Fluxionary hyperæmia in children.—The causes that produce it, as far as they are known, have been already briefly described. Bednar called attention to the fact that light attacks cannot be distinguished from the effects of a sudden rise in temperature. The fact that the symptoms are frequently observed in children when no fever is present, proves that they are not to be ascribed to fever alone.

In cases of intestinal irritation from indigestible food, and of severe catarrh of the small intestine, beginning with pain, and during the process of teething, phenomena evidently of cerebral origin appear, of which a partly hypothetical but not very satisfactory explanation has already been offered.

The children are usually restless, do not sleep well, cry in a complaining tone, and appear to be in pain. The eyes are generally kept closed, the pupils are slightly contracted, but react well. Headache is indicated by distortion of the features, elevation of the upper lip, wrinkling of the forehead, and spasm of the lids; the children refuse the breast, or nurse only for a short time. Sometimes, though rarely, the children are drowsier than usual, and must be awakened for nursing. Vomiting and constipation are frequent. Such is often the condition during teething, or in the beginning of an intestinal affection. If a free evacuation from the bowels takes place, all the nervous symptoms disappear. We repeat again that all this may occur without any elevation of temperature. If there be fever, its cause usually manifests itself soon (acute exanthemata, pneumonia, bronchitis, etc.).

An increase in the contents of the cranial cavity, sufficient to elevate the fontanelle, does not occur here; the pulse is usually quickened, varies according to the character of the fever, but is not irregular. The respiration is hurried, usually one-fourth as frequent as the pulse (Bednar). Slight convulsions frequently occur, such as trembling of the arms and legs, rolling of the eyes, either of each one separately, or of both together to one side or upwards, occasionally slight nystagmus, twitching of the face, or slight, clonic contractions of the diaphragm. The children are very irritable; they shrink from every noise and every touch, and cry piteously. Sudden eclamptic convulsions may follow these symptoms. These are difficult of explanation. When, however, they occur in weak children, during a severe attack of diarrhœa or of some other severe affection attended by fever, which is about to prove fatal, we are inclined to believe that the convulsions may be classed with those which are produced by closing of the venous canals (Hermann and Ganz). We have to deal here with diseases which are attended by rapid sinking of the heart's power; where the initial excitement of the heart is quickly followed by a paralysis. Under these circumstances, a point of time will come, when the whole column of blood in the brain will become stagnant, from the failure of the *vis a tergo*. As long now as the irritability of the central organ remains (but this period is always brief), convulsions will be caused by the lack of oxygen. This irritability of the nervous centres ceases as soon as the amount of carbonic acid in the blood passes a certain limit. We must not forget the fact, proved by experience, though difficult of explanation, that in childhood convulsions are much more readily and certainly produced by irritation of the nervous centres, than in adult life.

In other cases of eclampsia, however, the above conditions are not present, and the convulsions cannot be satisfactorily explained—unless we admit that a rapidly produced but temporary contraction of the cerebral vessels, consequent on irritation of peripheral nerves, is a sufficient cause.

It is evident from the above that the *diagnosis* of arterial fluxion in children is always difficult. It seemed easy when it was believed that we possessed in ophthalmoscopic examination of the retina a sure means of ascertaining the condition of the vessels in the pia and brain, but unfortunately the connection between the two localities does not seem to be complete. If the results of the ophthalmoscopic examination be taken as an indication of the state of the cerebral vessels, the idea of fluxionary hyperæmia will have to be entirely rejected. Consequently we are confined to a process of theoretical reasoning, which certainly, in a number of cases, where the above symptoms are present, will prove to be correct, but which is supported by no pathognomonic symptoms, and by but few well-founded physiological facts. Although it is true that the febrile cerebral congestion causes symptoms similar to those observed in other forms of like intensity, yet it cannot be admitted that the modern theory, that all the cerebral symptoms depend entirely upon the elevated temperature of the blood, and that the rapidity of the circulation, the calibre of the vessels, and the quantity of the blood contained in them, are of no importance, is sufficiently proved.

The *prognosis* of this condition in children is always doubtful. In the following chapter we will see that there is one affection, by no means as yet thoroughly understood, which is necessarily preceded by fluxionary hyperæmia. The so-called serous transudation of childhood (non-tubercular hydrocephalus acutus) is not unfrequently consecutive to cerebral hyperæmia, and is always fatal. We are not able to state, from our own experience, how often this is the case; Bednar states that out of every seven cases of hyperæmia one is followed by serious symptoms. This proportion is too high for children in general; however, it may be correct in the foundling institutions of large cities.

b. Fluxionary hyperæmia in adults.—The individual cases present great differences. It is only necessary to cast a glance over the description of the etiological influences, to see the reason of this. It is impossible to give a description of the symptoms which will cover all cases, and we will therefore content ourselves with describing a few of the most frequent types:

1. *Hysteria.*—Congestions are very frequent in the course of this neurosis, and are excited by the most diverse external causes.

The influence of the emotions of fear, anxiety, and sorrow, and of all sorts of peripheral pain, such as sudden and severe neuralgia (intercostal, facial), is especially evident. Intestinal affections, meteorism, constipation, are frequent causes. Often the most trifling peripheral pain is sufficient to call forth a sudden and severe congestion of the central organ. The individual predispositions, the increased facility with which reflex processes, both normal and abnormal, are excited, and the difficulty of controlling these processes in the cortex, cannot be too highly estimated. Under such circumstances the patient is seized with a sudden headache, which spreads rapidly over the whole head; there is a ringing in the ears, photophobia, a sensation as if the head would burst. He experiences diverse abnormal sensations in the periphery, alternating sensations of heat and cold, particularly in the trunk, tickling and formication of the extremities; nausea and vomiting occur; there is usually constipation, and the urine is very scanty. The symptoms may become worse, and a delirious condition be produced; the patient becomes incapable of thinking clearly and judging soundly; is irritable, sensitive, and quarrelsome. The face and conjunctiva are flushed; the pupils contracted, but react normally; the carotids pulsate strongly; usually the heart's action is increased in force and in rapidity, and every pulsation causes an exacerbation of pain in the head. In one case of well-marked hysteria, with paralysis of both extremities and a great variety of most remarkable nervous symptoms, the ophthalmoscopic examinations during the frequent attacks of severe cerebral congestion, regularly revealed a dilatation of the retinal arteries, so that the supposition of a transitory vaso-motor paralysis was justifiable. The condition was still further aggravated by violent palpitation of the heart. This was the only case of hysterical, cerebral congestion, in which we found the ophthalmoscope useful. Hysterical patients who never have severe cerebral symptoms are fortunate, for delirium frequently sets in, and may assume every possible character. Insane acts are not frequent, but a complete delirium called forth and kept up by hallucinations, particularly of hearing, may last as long as the congestion of the pia and brain, or even longer. There is no constancy in the form of the hallucinations; the delirium is often angry, sometimes sullen; sometimes there is complete confusion of ideas. The patient afterwards retains but an indistinct recollection of this state, like the impression left after dreams.

The *prognosis* is good; under proper treatment all the symptoms usually disappear in a few hours. Sometimes, however, remarkable and inexplicable nervous phenomena may follow. We have known it to be followed by catalepsy, lasting for whole days, and also by a prolonged state of deep hysterical sleep, with normal pulse, normal respiration, pallor of face, during which ophthalmoscopic examination showed the retinal vessels to be normal. Are we justified in assuming that there is œdema of the brain in these cases? This question cannot be answered.

2. *Chlorosis.*—A condition, similar to that in hysteria, occurs also occasionally in chlorosis, but the symptoms are not as severe. The consciousness is rarely lost, and the subjective symptoms retain their prominence. A singular form of chronic congestion is not infrequently met with. This hardly deserves the name of congestion; and we would not apply that term to it if dilatation of the vessels of the retina had not been observed

in one case. It occurs in females at puberty, sometimes also at a later period. In some of our cases an evident exciting cause existed (grief, some grievous loss, or a simple change of climate); in other cases the causes were to be found in the genital region, and could not therefore be clearly determined. The symptoms are: constant headache, usually moderate, but severe after violent movement or emotional excitement, and generally localized in the forehead; sleeplessness; impairment of the thinking faculty and of the memory, especially for circumstances of recent occurrence; a sustained train of thought can only be followed for a time with great effort, and its thread is then suddenly lost. All strong sensory impressions are exceedingly painful, and every intellectual effort increases the headache. The superficial vessels of the head are in a condition of chronic congestion. The menstrual flow is not usually interrupted, though frequently irregular. Sometimes, however, it ceases entirely. The functions of the stomach and intestines are usually disturbed; there are constipation and loss of appetite. The constipation cannot be permanently relieved, and its temporary relief does not improve the patient's condition much. The pulse is often small, and no over-activity of the heart can be discovered. At the same time the patient is often stout and well nourished, and, in consequence of the distention of the blood-vessels of the head, presents a blooming appearance, but she possesses little muscular strength, and is easily fatigued.

The disease is very chronic; it may last for half a year, and defy all treatment. Fortunately it often disappears of itself in time.

3. *Emotional excitement.*—The effects of these are very different in persons of different dispositions and degrees of excitability. Momentary loss of consciousness (syncope) is frequently caused by a sudden and unexpected emotional impression, especially one of a depressing character. Slight convulsive movements have been observed during the syncope, or it may be followed by an epileptic attack (fear). This is doubtless due to the violent initial contraction of the vessels of the brain which lasts an unusually long time (see above). If the emotional impression is not received suddenly, it may throw predisposed per-

sons into a condition of restlessness, sleeplessness, and excitement with headache, and all the signs of a severe cerebral irritation, which can only be explained by the supposition of a transitory fluxion.

A case of this sort came under our observation: A woman, after the death of her husband, became delirious and remained so for ten hours; the delirium was of a happy character, and was unattended by fever; all the signs of an intense cerebral congestion were present.

4. *Febrile processes.*—The cerebral symptoms in fever are attributed by some to the altered condition of the blood, by others to the disturbance of the circulation in the brain. Both, doubtless, are influential, but it is difficult to determine the exact share of each in the production of the symptoms.

Some persons can bear no emotional excitement, while in others the slightest fever causes marked disturbances of the cerebral functions, the most common being somnolence, loss of memory, confusion of ideas, and impairment of the judgment. The headache, giddiness, and elevated temperature suggest a commencing cerebral affection; but all the cerebral symptoms disappear, and the cause of the fever assumes its proper prominence, as soon as the temperature falls. If the temperature continues high, a certain tolerance seems, in time, to be established. In such cases it may be fairly said that the symptoms are due to the elevated temperature alone. In other cases, however, the evident signs of congestion of the head are present, such as headache, dizziness, extreme faintness, ringing in the ears, flashes of light, deceptive sensory impressions; in consequence of this last the patient forms wrong judgments, and performs wayward acts (he wants to rise, and to go out, resists supposed injuries, etc.). Such a state may be produced by congestion, in susceptible persons, even when there is no fever. The symptoms increase or diminish in violence with the intensity of the congestion.

Consequently there undoubtedly are cases in which all the symptoms depend upon the elevation of the temperature, while in others they are due to the congestion. Liebermeister's theory is best illustrated in typhoid fever; but even the little that we know about the alterations of the cortical substance in typhoid proves that there is something else behind the typhoid affection of the brain.

Chill.—A violent congestion of the inner organs occurs during a chill; it is well known that the temperature of the body rises greatly when the vessels and muscular elements of the skin become contracted. An explanation of the mechanism of this hardly falls within the limits of this paper. On the other hand, the question should be decided as to how much of the transitory, intense delirium of intermittent fever is due to the high temperature and how much to the collateral cerebral congestion. This is a difficult point to decide with any degree of certainty, owing to the different temperaments of different individuals. In some the symptoms appear when the elevation of temperature is but moderate, while in others no cerebral disturbances are caused by a hyperpyrexia of short duration. The subjective symptoms of the cerebral fluxion are headache, giddiness, ringing in the ears, flashes of light before the eyes; in some individuals there are hallucinations, and delirium which is sometimes very confused, sometimes assumes a specific character (fear of death, aggressive actions, etc.). These symptoms do not disappear after the cold stage is passed, but they last till the febrile and the sweating stage, and in some cases even continue after the attack is entirely over. Here, too, we do not doubt the existence of anatomical changes as yet undiscovered.

5. *Delirium of poisoning.*—That there is congestion in this affection is unquestionable; but here, too, we cannot prove that this congestion is the cause of the delirium. The usual symptoms are irritability alternating with drowsiness, headache, giddiness, temporary excitations of the sensory and motor centres. We would disregard known facts if we should attribute the powerful effects of certain poisons (alcohol, atropine, nitrite of amyl) upon the psychical functions, entirely to the cerebral congestion, especially as it is still doubtful whether all of these drugs produce congestion. We need not stop here to describe the delirium produced by alcohol or atropia.

6. *Hypertrophy of the heart.*—Here we have a permanent increase in the power of the heart, and at the same time a second-

ary dilatation of the vessels; consequently two causes of congestion in constant action. However, the degree of vascular distention. will rise and fall according to the force of the heart's action, which, in these cases, as is well known, is subject to many changes; the fluxions therefore vary greatly in intensity.

Patients suffering from this affection complain almost constantly of headache and a sense of pulsation in the head (though many of them in time become accustomed to the abnormal state). They have hot, flushed faces, and complain of disturbed sleep and uneasy dreams, giddiness, and numbness. The throbbing in the head is increased by exertion, and may be relieved at times by copious epistaxis. A slowly developing degeneration of the vessels of the brain may set in, and lead to extensive apoplexy with all its results (cirrhosis of the kidney). All this, however, lasts only for a certain variable length of time; very different symptoms are experienced as soon as the unavoidable degeneration of the heart becomes marked. Then the pulse wave becomes small, and a condition of arterial anæmia of the brain follows, accompanied by attacks of syncope, etc. In well-marked cases a dilatation of the retinal arteries with capillary pulse is seen.

Similar phenomena are to be observed in morbus Basedowii. The varied nervous symptoms presented in this disease attracted the attention of the earliest observers. Great irritability, anxiety, a feeling of oppression, headache, vertigo, ringing in the ears, sleeplessness, restlessness at night, hurried movements (extreme hilarity), are rarely wanting. Here also we have found dilatation of the retinal vessels and capillary pulse.

7. *Inflammations about the head.*—Erysipelas may be taken as the prototype of this class. Headache, mental confusion, excitement, anxiety, photophobia, are the symptoms of the cerebral fluxion usually observed, and they may become very intense. Delirium of every variety and every degree of intensity may occur, and the restlessness may gradually change into coma, while the temperature often ranges quite high. Some may attribute this termination to the elevated temperature alone; usually cerebral hyperæmia is the only lesion found, but we are con-

vinced that accurate observation will in the future demonstrate more essential anatomical changes.[1]

The congestion of erysipelas does not terminate in a purulent meningitis. The meningitis of the convexity, which not infrequently complicates erysipelas, we believe to be a metastasis, and have only observed it where there was suppuration.

These few examples certainly do not exhaust the subject. Congestion of the brain following a cold bath, or after exertion, could also be described. The mode of its production will be understood if the influence of increased force of the heart's action, of reflex or paralytic dilatation of the vessels, and of collateral fluxion, be kept in mind. The ground on which the whole structure is based is, however, uncertain, and hence the description given above is rather fragmentary.

c. Hyperæmia in old age.—It has been customary for observers, following the lead of Durand-Fardel and other French authors, to include many of the conditions frequently met with in old age under the head of congestion of the pia and brain. The coincidence of congestion with the senile changes in the vessels and the heart is unquestionable, but it is very doubtful whether the symptoms of impaired intelligence even when transitory are always accompanied by congestion. Durand-Fardel divides the affections into two groups—the first characterized by torpor, by decadence of the functions; the second by symptoms of cerebral irritation. The first group includes an apoplectic and a sub-apoplectic form, which differ only in degree; the second, the acute delirium of old age and convulsive attacks.

First of all, it must be premised that sudden attacks of acute fluxion may arise in old age, under the same circumstances as in youth; but these are modified somewhat by the alterations that have taken place in the circulatory system. It has already been pointed out that in addition to the increase in the amount of blood in the brain or in particular parts of it, its circulation is frequently retarded. This changes the aspect of affairs essentially, for the increased amount of blood will be changed more slowly than under similar circumstances in younger persons, and hence there is necessarily an alteration in its quality; it resembles venous blood more closely. This may greatly modify the symptoms, if the affection has attained a high grade. Frequently, too, anæmia of portions of the brain accompanies hyperæmia of other portions.

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[1] In a case where death occurred with the above-mentioned symptoms, the pia was perfectly normal, but migration and a small extravasation of white blood-corpuscles were found in the white substance of the hemispheres, exactly like what is seen in certain stages of dementia paralytica.

Thrombosis of a diseased vessel by which the supply of blood is cut off from the part supplied by it seems to be much more frequent in the senile brain than is usually supposed. Whether a sufficient collateral circulation will be established or not, will then depend upon the locality affected. If a thrombus forms in the small branches of the arteria fossæ Sylvii or corporis callosi, which supply the ganglia, the collateral circulation will be much less readily set up, especially when the vessels are diseased and wanting in elasticity, and the *vis a tergo* is diminished, than when an artery of the pia is affected. If collateral circulation be not established, a spot of necrotic softening will be developed, as after embolism. The symptoms will vary with its position and extent, and a sufficiently accurate localization of the disease may be made from them. Elsewhere such an occurrence would not exert much influence upon the surrounding tissues; but in the brain it must be regarded as an irritant, which leads to increased arterial congestion, provided the condition of the heart at the time will permit it. In this way it may happen that while the greater part of the brain is in a state of arterial hyperæmia, the ganglionic tract of one or rarely of both sides is fatally anæmic. If one side only is affected, a transitory or permanent (according to the condition of the circulation in the interior of the brain) paralysis may be added to the symptoms of congestion.

In cases of sudden coma or convulsions, we are not justified in concluding that the sole cause is hyperæmia, even if the congestion of the external vessels be well marked. In the case of one old man, who was suddenly seized with all the symptoms of Durand-Fardel's coup de sang, such as all the signs of great external hyperæmia, stertorous respiration, and slight convulsions, very great anæmia of the crura cerebri and medulla was found, and no decided hyperæmia of the cortex and the corona radiata. The cause was an old thrombus in one vertebral artery, which had extended into the basilar artery. In consequence of extensive atheroma of the carotids and the circle of Willis, and of the condition of the heart, a rapid change in the direction of the blood-current was impossible, and the result was instantaneous anæmia of the posterior part of the crura cerebri from the corpora quadrigemina forwards; this caused labored respiration, then convulsions, and finally rapid paralysis of the respiratory and circulatory centres, thus affording a good illustration of Nothnagel's convulsion centre. Such cases are calculated to produce the greatest skepticism as to the connection of coup de sang with the convulsions of old age. This example illustrates the fact that, in the majority of cases, the condition is a complicated one. The existence of congestion cannot be denied, and the conditions of slighter psychical excitement may be correctly attributed to it, yet, in many cases, its importance is decidedly secondary. Anæmia of certain parts of the brain, in the attempt to compensate for which, great arterial congestion of other portions may be produced, will cause symptoms of increased excitability in one portion of the nervous substance (medulla oblongata), and of rapidly diminishing excitability in others (the hemispheres). The retardation of the circulation, too, will cause some special symptoms, varying according to the locality in which the change occurs. When the cortex is the part affected, its functions are not impaired in any definite way (the somnolence which is so frequent cannot be with certainty attributed to it); when the ganglionic region is affected, the conducting power of the bands of fibres passing through it will be impaired; when the centres of the medulla are involved, symptoms of increased excitement will ensue, followed by enfeeblement, and finally death. The apoplectiform symptoms, therefore, which are usually comprised under the term coup de sang, require a careful analysis in every case, and in some cases we will succeed in finding an explanation for them. The history of an attack is as follows: Usually symptoms, due to a simple congestion, precede the attack, viz.: dizziness, dull headache, ringing in the ears, irritation of the central portions of the nerves of sense, heaviness, and numbness of the limbs, specks before the eyes, occasionally muscular twitchings with slowness in performing the voluntary movements, and diminution of the capacity for sustained and acute thought. In addition to these, there are the objective signs of external congestion, redness of the face, pulsating carotids, and palpitation of the heart. Usually there is nausea, or vomiting, and constipation. Everything which increases the force of the heart's action aggravates these symptoms. Suddenly an aggravation of all the symptoms sets in. There are severe palpitation, strong carotid pulsation, and redness of the face; a burning sensation in the head is complained of, and confusion of thoughts and finally loss of consciousness occur, more or less suddenly. The loss of consciousness is sometimes complete, sometimes incomplete. Often the patient retains an indistinct recollection of what happened. In some cases there is moderate contraction of the muscles, rarely partial convulsions of one or more extremities; sometimes the muscles are perfectly relaxed, and there is no motion. A transitory hemiparesis, or facial paralysis, is frequently observed. If the patient recover after a short time, the hemiparesis may continue for a time; often it disappears entirely in a few hours, but it may last for some days and disappear gradually. Death sometimes takes place during an attack; it is preceded by stertorous respiration, small, quick, frequently irregular pulse, and occasionally by convulsions, and finally complete paralysis of the heart sets in. These are the cases in which a careful anatomical examination will certainly reveal the existence of some other cause of death than simple cerebral hyperæmia. It was in an autopsy on one of these cases that the lesions described above were found. The symptoms are really those of anæmia of the crura cerebri; the hyperæmia of the pia and the cortex we will not take into consideration.[1]

The same can be said of Durand-Fardel's convulsive form of congestion; moreover, he himself confesses that it cannot be sharply differentiated from the apoplectiform variety, because the symptoms of the two forms are intertwined in so many different ways. The convulsions resemble those of epilepsy, but are without the initial cry and the long sopor. Of course nothing can be settled by such slight differences. According to Durand-Fardel, the attacks are occasionally accompanied by fever. Undoubtedly much heterogeneous material has been grouped under this one head, but it can hardly be believed that we have to deal in these cases with simple senescence of the brain. We have pointed out above a more probable explanation of the symptoms.

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[1] When we glance over the recorded cases—particularly those of Durand-Fardel's—we cannot resist the impression that not a few of them (especially tho-e which presented extreme pallor of the face) were instances of simple cardiac paralysis in old people previously sick.

Finally, the transitory acute delirium, which is not rare in the cerebral atrophy of old age, is also attributed to intense hyperæmia of the brain. This may be true, but not in all cases, as this delirium frequently occurs, when not even the slightest evidence of congestion can be discovered. The delirium usually breaks out suddenly, often at night. The patient becomes excited, no longer knows where he is, does not recognize his attendants, breaks out into fits of motiveless anger, and scolds, storms at, and attacks those around him. His reasoning is entirely illogical and incoherent. Occasionally the patient recognizes those around him, but is unreasonably angry with them. He performs the most wayward acts, is unwilling to remain in bed, practises all sorts of foolish acts with lifeless objects, and moves his arms about, reaching after imaginary objects. There is frequently an impediment of speech, very like aphasia; the patient makes great efforts to speak, but cannot find the proper words. There is never headache during the attack, and we have never seen it accompanied by fever; the tongue is usually dry. Durand-Fardel describes a striking symptom (which is not, however, always present), namely, a sero-mucous secretion from the conjunctiva and the mucous membrane of the mouth, which disappears immediately on cessation of the delirium. There is no vomiting, but constipation is usually present.

It is impossible to point out the pathological process in the brain, in cases of this sort in which congestive changes are certainly absent. The sudden outbreak of hallucinations of sight or hearing seems to be sometimes the only exciting cause, just as it is the cause of many maniacal attacks among the insane. This, however, is not true of all cases.

d. Venous hyperæmia.—The symptoms and the anatomical results are more striking when the cause of the venous stasis is situated near the capillary system of the brain. Thrombosis of the sinuses is of the very greatest importance on account of its effect upon the circulation of the cortex; no sufficient collateral circulation can be established, because the existing collateral vessels are too few in number.

When the cause is situated lower down, so that the blood, in addition to the collateral vessels within the cranium, can also make use of those outside of the skull (compression or thrombosis of one vena jugularis), the stasis cannot attain a very high grade, and the symptoms are not very severe: headache, some somnolence, indolence of intellect, and loss of excitability. When the cause is situated in the thoracic organs, all the collateral vessels will be dilated (including the thyroid veins), and the pressure of the retarded blood current is then distributed in so many directions, that a dangerous degree of hyperæmia of the brain cannot take place. Hence, even the great congestion that occurs during the later stages of diseases of the lungs and the heart, do not excite very severe symptoms (heaviness and pressure in the head, drowsiness and dullness, pain, diminution of intellectual activity). However, the diminution in the amount of blood, due to the impaired nutrition in these cases, must also be borne in mind. We might be tempted to attribute many of the sudden attacks of dyspnœa occurring in cases where the general circulation is retarded, and the blood has accumulated in the veins (degeneration of the heart), to a lack of oxygen in the blood in the brain; the causes of this dyspnœa are however usually to be found in the state of the lungs. The final symptoms of affections of the heart are due to weakness of that organ and its consequent inability to supply the brain with enough blood that has undergone the necessary oxidation in the lungs.

Prognosis.

The prognosis requires but a brief consideration. We have already said all that is necessary with regard to the prognosis of the hyperæmias of childhood. In adults the different forms must be considered separately. The congestions of hysteria are transitory, and are merely of importance from the fact that, after they once make their appearance, they are very liable to recur.

The prognosis of the chronic hyperæmia of chlorosis is good, for the greater number of the cases usually get well spontaneously; the disease however lasts a long time, and therapeutics do not afford very satisfactory results. In all the other forms of cerebral hyperæmia the duration and termination are dependent upon the primary disease. The congestion caused by cardiac hypertrophy cannot be cured, but its subjective symptoms may be relieved by treatment.

True fluxion is ominous when the cerebral vessels are diseased, there being always danger of rupture. The acute delirium described above is in itself of little significance, but its causative lesions are incurable.

It is impossible to say anything more precise about the prognosis of the coup de sang than is contained in the preceding pages, because so many different anatomical processes are concerned in its production.

Treatment.

An accurate description of the therapeutic measures to be employed in each special variety of congestion, would carry us far beyond the limits of our task. It would involve the consideration of a number of the diseases of children, the anæmia of adults, hysteria, senile atrophy of the brain, etc. We will, therefore, only call attention here to a few practically important points.

The following are the measures that may be employed during an attack:

a. Venesection.—This can be used with great advantage in stout, healthy persons of either sex, whenever there are evident signs of severe cerebral fluxion—especially when there is violent action of the heart, with carotid pulsation and a full, tense pulse.

Of course, the nutrition and strength of the patient are to be duly considered, to decide whether the patient is in a condition to bear a venesection without injury.

This consideration will sometimes lead us to abstain from blood-letting, when it is apparently indicated, and the milder means are then often effectual. We must be particularly cautious when we have to deal with old persons, whose arteries are rigid, and always bear in mind that in these cases the existence of hyperæmia of the tissue of the brain is by no means certain even when the manifest symptoms of congestion are present. Venesection is rarely to be thought of in venous hyperæmia. If dangerous symptoms be present, they are due to the feebleness of the heart.

b. Local abstraction of blood.—This may always be employed, and is allowable, with proper caution, even when the diagnosis is uncertain. Leeches to the temples and behind the ears (from six to eight in number; as many as thirty or forty were often applied by the older practitioners), and cups to the neck and back are to be tried.

c. Cold.—The application of ice-bags to the head should never be neglected. The cold, however, must be energetically used, else the method is worthless.

d. Derivatives to the skin.—Junod's cupping boot is very useful, if it be at hand; also partial baths, to dilate the peripheral vessels (arms, feet, legs), and the application of warm fomentations to the extremities or the body, of hydropathic packing, and mustard plasters to the extremities. Blisters work too slowly to be of use; they are of the least benefit when applied to the head or neck.

e. Derivatives to the intestine.—In using the favorite calomel, care must be taken to obtain the requisite action. The saline cathartics are preferable, and the stronger laxative mineral waters, which work quickly when warmed. If these fail, castor-oil or infusion of senna may be used. The action of laxatives may be assisted by clysters.

f. Rest and *careful diet* are, of course, essential. Drinks containing carbonic acid are to be avoided.

In small children all these measures must be modified to suit their more susceptible constitutions.

Other modifications readily suggest themselves. All depressing agents must be avoided in the treatment of congestion in chlorotic or hysterical persons; in the latter the expectant plan is the one that every practitioner will finally adopt. The chronic cerebral hyperæmia of chlorosis demands the most careful treatment. Abstraction of blood is of no use, and is injurious if carried to any extent; only temporary benefit can be expected from derivatives to the skin and the intestinal canal. Occasionally the saline mineral waters, drunk at the spring, prove useful. Preparations of iron and the ferruginous waters are very frequently of no benefit, and may even be injurious. An entire change in the mode of life, together with a change of climate (residence in the mountains or at the sea-side for some months), has in our experience given the best results of all the methods of treatment. Some obstinate cases have yielded to galvanization of the head and of the sympathetic nerve in the neck; but in the greater number of cases no benefit was derived from this treatment. When fever is present an attempt should be made to distinguish between the effects due to congestion and those due to the elevated temperature. When there is violent congestion, this should be reduced by the use of the measures enumerated above, before the application of cold is resorted to. On the other hand, an energetic antipyretic treatment will produce the desired result, when the symptoms of abnormal cerebral action are present without severe congestion.

The physician often finds himself in a dilemma where the heart is hypertrophied; he must decide whether it is more advisable to depress the action of the heart, in order to diminish the cerebral fluxion, or to bear with the latter rather than cause a state of enfeebled circulation. In certain stages of aortic insufficiency and of cirrhosis of the kidney, the possession of great cardiac power is essential to the well-being of the patient. In such cases we should endeavor to relieve the subjective symptoms by the use of cold and small doses of narcotics.

B.—Acute Inflammations of the Pia Mater.

A division according to etiology would be the most satisfactory in this group of diseases, as well as in all other domains of pathology; but as our knowledge of them is too limited to permit of this, physicians have constantly sought to establish a nomenclature and classification based upon the anatomical changes.

It was unfortunate for the development of the knowledge of these affections, that almost all of them, in consequence of the dilatation of the ventricles and the increase of the ventricular fluid, were classed by observers, until quite recently, as forms of hydrocephalus. This view has been detrimental in many ways. It has led to a confusion of the clinically much less important chronic hydrops cerebri, which is developed often very slowly, as a result of many atrophic processes in the brain, with evidently inflammatory affections possessing similar, gross, anatomical lesions. The dividing line cannot even yet be sharply drawn, as the anatomical basis is in some respects still inaccurate.

The subject is still more complicated by the resemblance of the changes in the pia resulting from the development of miliary tubercles, to those caused by a simple inflammatory effusion. At one time the latter were almost universally classed as tubercular meningites. Finally the evident differences of the processes in adults from those in children add to the confusion. On this account some authorities, who have not had many opportunities for making autopsies on children, have doubted the occurrence of certain forms of the affection during childhood.

At the present time we are unable to classify these affections according to their etiology, because we do not yet know the causes of many of them. We know indeed the circumstances under which they arise, we are acquainted to a considerable extent with the nature of the anatomical processes, but the final, real cause is still enveloped in mystery. We base our present classification upon the following points:

I. There is an inflammatory affection of the pia in childhood the special nature of which is still uncertain. It causes a more or less rapidly increasing effusion into and dilatation of the ventricles. It has no connection with tuberculosis, no traces of miliary granulations being discoverable. The demonstration of its inflammatory origin is difficult, but nevertheless we will attempt to prove it later on. This is the non-tubercular hydrocephalus acutus of children.

The term hydrocephalus will be dropped, as it only designates the result of an anatomical change, and in its stead the affection will be called *leptomeningitis infantum*, a name which may be considered to be a provisional makeshift.

This condition is extremely rare in adults.

II. Entirely distinct from this affection is the *tubercular meningitis* (basilar meningitis, or hydrocephalus acutus, a name frequently given to it as well as to the previous variety), an inflammatory affection dependent on the development of miliary tubercles in the tissue of the pia. This may occur in almost all ages. Its anatomical components are as follows:

a. Miliary tubercles in the pia varying in number and especially in age.

b. Inflammation of the pia usually most marked at the base, but not constant even there. The inflammatory appearances in the pia often very slight.

c. The hydrocephalic effusion—not always, however, present.

d. A consecutive affection of the cortex. The etiology of this affection can fortunately be more accurately described.

III. Leptomeningitis with pus, evident to the naked eye, and without miliary granulations. The purulent deposits are conspicuous and are found in different parts of the pia. Not unfrequently there is also a considerable hydrocephalic effusion. This meningitis of the convexity, as it is often incorrectly called (the convexity is by no means the only part affected), includes a number of pathological processes that differ in their etiology. As, however, our knowledge is still insufficient for an etiological classification, we are compelled to make the following subdivisions:

1. *Basilar meningitis with great ventricular effusion*—a rare affection, occurring in adults and young persons; chronic in its course, its etiology still enveloped in darkness.

2. *Spontaneous purulent meningitis of the convexity*—called spontaneous because its cause is unknown.

3. *Traumatic meningitis.*

4. *Meningitis due to the extension of suppurative processes from neighboring tissues to the membranes of the brain* (meningitis consecutive to caries of the cranial bones, particularly of the petrous portion of the temporal, etc.).

5. *Metastatic meningitis*, *i.e.*, that form which accompanies suppurative processes at a distance (peritonitis, puerperal fever, pericarditis, pneumonia, erysipelas, caseous pneumonia with purulent liquefaction, dysentery, typhoid, acute exanthemata, pyæmia, pleurisy, endocarditis, diphtheria).

6. *Epidemic cerebro-spinal meningitis.* This, being a contagious disease, is not to be considered here.

Effusion into the ventricles occurs in all of the above-mentioned forms, but is constant only in the first, and therefore cannot be considered a proper basis for classification. In contrast with these various forms of inflammation of the pia, the existence of an inflammation of the ependyma ventriculorum is exceedingly doubtful. No anatomical evidence of its existence can be obtained. We will consider this point more fully in the proper place.

Affections of the cortex very frequently accompany the meningitic processes, but only a small number of them are accurately understood. If the close vascular connection between the brain and pia be kept in view, it will not seem strange that changes in the brain should follow disease of the pia.

The visceral layer of the arachnoid can no longer be described as an independent structure; for our purposes it may be regarded as the outermost, somewhat thickened layer of the pia. Consequently we no longer speak of a subarachnoid space, but describe the numerous more or less extensive spaces which are found, wherever the arachnoid is connected with the pia by connective tissue septa, as meshes or spaces in the tissue of the pia (subpial space). The largest of these are situated at the base of the brain, and are directly continuous with the corresponding space around the spinal cord; the smaller ones are situated on the convexity and communicate with each other, but all of them do not communicate with the large spaces at the base. These structures have no connection with the ependyma of the ventricles.

The pia dips down into all the irregularities on the surface of the brain, its external layer does not accompany it in these excursions, but is stretched across the sulci and depressions like a bridge.

The pia is prolonged into the interior of the brain only at the scissura transversa cerebri, between the splenium corporis callosi and the corpora quadrigemina, and also along the entire length of the fissure by means of which the middle cornu is opened on the side of the pedunculus nearly to the extremity of the gyrus uncinatus. The membrane enters the fissures on both sides, but loses immediately the character of a double membrane; it forms all the vascular plexuses of the ventricles. The pia is mainly composed of vessels; the connective tissue is not abundant, and in places, where the vessels are especially numerous, it loses its fibrous appearance almost entirely. The plexuses contain a much larger number of exceedingly convoluted vessels. In mammals and in the human embryo they are covered by ciliated epithelium, supported by a very thin layer of connective tissue, which, however, often entirely disappears, so that the vessels alone support the epithelium. The connective tissue between the vessels is very delicate, and in no place distinctly fibrillar.

The arterial supply of the pia is mainly derived from the basilar artery and the circle of Willis. The anastomoses, both arterial and venous, are very complete; it possesses a special capillary network.

Branches from the arteries of the pia enter the cortex; they are small, penetrate perpendicularly into the brain, and branch into a delicate but relatively wide-meshed capillary network. The small calibre of the branches to the cortex, as well as the fact that large extensile spaces exist in the pia, certainly lessen the influence of disturbances of the circulation in the pia upon the cortex.

The pia possesses many nerves, but unfortunately their mode of termination is still unknown. They are generally thought to be vaso-motor nerves. Kölliker has followed them into the brain as far as to vessels only 0.09 mm. in diameter. The plexuses are said to have no nerves. The nerves are derived in part from the sympathetic (carotid, and vertebral plexuses), in part from the roots of the cerebral nerves, and, finally, delicate fibres are said to enter the pia directly from the pedunculus and the medulla oblongata. The pia, nevertheless, is said not to be sensitive. Finally, the extraordinary richness of the pia in lymphatic vessels greatly favors the production of pathological processes. These vessels are connected with the perivascular lymph spaces of the brain, and consequently when they are filled with pathological products or are obliterated by compression, an obstruction to the plasmatic current out of the brain is produced. The existence of an epicerebral lymph sac is not now admitted.

I.—*Leptomeningitis Infantum* (*Hydrocephalus Acutus sine Tuberculis*).

This term was chosen partly to avoid the necessity of employing the name hydrocephalus acutus, and partly because we are convinced that in a certain number of the cases a true inflammatory disturbance exists.

When we examine the literature of this subject, we find everywhere a lamentable and exceedingly confusing discrepancy of views. This seems to us to be due especially to the fact, that too little attention has been directed to the etiology of the affection. There is no doubt that conditions of a very different character *may ultimately produce lesions exactly alike in gross anatomical appearances or only differing in unimportant details.* This does not, however, justify us in classing the different processes together. We do not consider it correct to say (as Niemeyer does) that a hydrocephalic effusion can be caused by increased pressure in the vessels, by an abnormal quality of the blood, or finally by disturbances of nutrition which make the walls of the vessels permeable, and that the changes resulting from all these different causes are to be regarded as one disease; or to classify the cerebral effusion, which is due to diseases of the kidney and general hydrops, with that which is dependent upon the intense fluxionary hyperæmia of childhood during severe eruptive fevers (Rilliet and Barthez, Leubuscher); or to hold that there is no difference between the effusions occurring in atrophic children who are suffering from chronic affections of the digestive organs and all other forms of hydrocephalus.

It is evident, then, that the etiological influences demand a primary consideration. Acute effusions into the ventricles occur during childhood:

1. As the result of a variety of fluxions to the brain and pia. We must refer here to the discussion of this subject in a previous chapter, which was undertaken solely with the view of obtaining a satisfactory basis for the subsequent discussion of the inflammatory processes. It is true that we have not yet discovered the bridge which connects simple hyperæmia with inflammation, and that we know of no specific provocation, such as we find in meningitis tuberculosa, for instance; but still we will find that there is evidence to prove that we have to deal with an inflammation of the pia, which assumes its peculiar character in consequence of its development in the infantile brain.

2. Hydrocephalic effusions take place in all affections attended by general hydrops; the most common cause of hydrops in childhood is scarlatina. A distinction must of course be made between the effusions which occur during the eruptive fever, and which fall under the first category, and those which occur at a later period, during the existence of the scarlatinal nephritis. The latter are not very rare in children, but are very rare in adults. The opinion that we have to deal here with an active process in the brain, has long been discarded. Moreover, a certain number of the cases which are called hydrocephalus intra vitam are really cases of uræmic intoxication (Odier, Rilliet), as careful examinations of the urine would easily prove. In spite, however, of this absolute etiological discrepancy, the hydrops of nephritis scarlatinosa is still often held to be a cause of acute hydrocephalic effusions of an "active" nature.

3. Effusions into the ventricles also occur in marasmic conditions, especially those which depend upon chronic affections of the digestive apparatus. It is evident that the remarkable similarity of the symptoms arising from an anæmic state of the

brain in children with those due to a hyperæmic condition, will often render an accurate differentiation impossible, in spite of the well-known clinical picture of hydrocephaloid described years ago by Marshall Hall. The variability of these symptoms is probably the cause of the frequent mistakes. The fluid in these cases is, moreover, not effused in large quantity; it is partly a hydrocephalus ex vacuo, in consequence of the diminution of the volume of the brain (sunken fontanelles, overlapping cranial bones), partly the result of the long-continued weakness of the heart's action, and slowness of the circulation, with greater emptiness of the arteries than of the veins.

4. Effusions may also follow any affection of the brain which causes disturbances in its circulation (tumors of every variety, abscesses of the brain, etc.).

5. Hydrocephalus may also result from venous stases, particularly those forms in which the composition of the blood is at the same time altered to a considerable extent (lesions of the heart, emphysema, ulcerative affections of the lungs, caseous infiltration, or impeded respiration from deformities of the thorax, such as rachitis and spinal curvature).

6. Finally, it may result from local obstructions to the escape of the blood from the cranium (tumors near the large veins of the neck, enlargements of the thymus).

Inasmuch as in many cases effusions from various causes are commingled, it will be easily understood that in such cases it is impossible to attain perfect clearness of description. The above causes alone, however, are those with which we have to deal here.

Any one who has opportunity to observe a number of cases will be convinced that every cerebral fluxion in childhood, which is of long duration, and of a certain intensity (varying in different constitutions), may lead to serous effusion into the ventricles. We are forced to assume a true inflammatory process as the cause of this effusion, but at the same time it is impossible to say, during life, when the hyperæmia ceases and the inflammation begins. It is also possible that every hyperæmia of the brain in childhood causes a certain amount of effusion, which, however, is reabsorbed in the majority of the cases.

The affection is most common in children between one and five years of age, but it is sometimes met with both in younger and in older children. The statement is often made that a certain predisposition, a peculiar condition of the nutrition, must have pre-existed in the cases in which a simple fluxionary hyperæmia has terminated fatally. It must be confessed that only the minority of the children affected were perfectly robust and free from acute or chronic disease previous to the cerebral disease.

Still we meet with a much larger number of cases than in meningitis tuberculosa, in which the constitution was previously perfectly intact. A state of impaired nutrition, a bad constitution, or the pre-existence of rachitis or scrofula, is, consequently, by no means a conditio sine qua non. A small majority (accurate figures cannot be given) of the children certainly present considerable emaciation, and a retarded development of the body, which can usually be traced to some special, general affection, or some severe, acute disease, as rachitis, scrofulosis, disease of the lungs, or an acute exanthema. The affection may follow bronchitis, pneumonia, measles, or pertussis, although the antecedent disease itself furnishes no provocation for the production of the new cerebral disease. Children who are subject to frequent attacks of hyperæmia of the brain, or who have suffered from convulsions, the causes of which were obscure, are more liable to the disease. Hereditary influence seems to be of much less importance here than in meningitis tuberculosa—at least the existing evidence in regard to this matter fails to show the connection. Both sexes seem to be about equally affected. The influence of locality and of the conditions of life is evident. Cities and foundling-asylums furnish a large number of cases. The disease is less common among the well-to-do country people.

In the present state of our knowledge, it is hardly worth while to devote much attention to this "predisposition" to the disease.

The following are the exciting causes of the affection:

1. Cerebral hyperæmia in dentition. (Compare the previous remarks on the occurrence of hyperæmia in such cases.)

2. Cerebral hyperæmia in eruptive fevers (measles, scarlatina).

3. The severe febrile condition of acute pulmonary affections. We have never met such cases.

4. Violent concussion of the cranium. This is mentioned, and its influence, *à priori*, cannot be denied, but we have never met with an instance of it.

5. Certain authors report having seen the affection break out in the course of severe intestinal catarrhs, and the statement is given here for the sake of completeness.

6. Alcohol. Goll has made some interesting observations on this point, which, unfortunately, he has never published. He several times saw symptoms set in in children, after the ingestion of wine, which could arise from nothing else than a hydrocephalic effusion.

The following is a typical case:

8. Female child of eleven months; previously perfectly healthy, excepting occasional disturbances of digestion; well nourished; no enlarged glands; no hereditary disease; no rachitis. A brother and sister are at present suffering from measles. The child was taken ill with restlessness, thirst, fever, 39.8° (103.6° F.), loss of appetite; the conjunctivæ were injected, and there was a sero-mucous discharge from the nose; redness of the mucous membrane of the mouth and pharynx; a few aphthous ulcerations on the tongue. (First day.) At night restlessness, tossing about in bed; constant outcry; high fever in the morning; pulse 130, regular and somewhat full; conjunctivæ and face flushed; increased discharge from the nose; much cough; numerous dry râles heard over the lungs, in front and behind; temperature 39° (102.2° F.) during the whole day; constipation; no important nervous symptoms. (Second day.) Night similar to the preceding; vomiting in the morning; everything that is taken is rejected, even water; constipation; meteorism of the abdomen; injection of the conjunctivæ and the face the same; discharge from nose more purulent; pharynx and larynx free, but more cough. and more dry râles; temperature above 39° (102.2° F.) during the whole day; pulse 140, and regular. Towards evening the child became more quiet; she lies usually on the side; groans frequently; keeps her eyes usually convulsively closed; mouth is drawn, as if in pain; more vomiting, and a slight passage from the bowels after a laxative; fontanelle pulsates forcibly, and begins to arch upwards; pupils are rather small, react slowly; no strabismus. Towards evening a slight convulsion of the whole left side, excepting the face; late in the evening the eruption of measles was seen on the face and breast. (Third day.) No convulsion during the night, but vomiting; no operation, although a cathartic was given; less restlessness; patient lay motionless for hours; occasional cry; increase of reflex irritability, sudden starts. Temperature 39.5° (103.1° F.) in the morning; eruption has not increased, is limited to the neck, breast, and lower part of the face; narrow pupils, which

react very little; no strabismus; no difference in the contraction of the muscles of the face and the extremities on both sides; pulse 140, irregular; vomiting; constipation; abdomen slightly distended; no stiffness of the neck or of the peripheral muscles; fontanelle more arched, but pulsations feebler. In the course of the morning a violent eclamptic convulsion occurred; tetanus of the neck, opisthotonos, convulsions of the arms and legs; spasm of the muscles of inspiration, until complete cyanosis was produced; pupils dilated, but varying constantly in their size; convulsions became more clonic after a few minutes; spasms of the inspiratory and expiratory muscles, and twitching of all the extremities. Attack lasted about ten minutes, and was followed by sopor with half-closed eyes, contracted, nearly motionless pupils, and relaxation of the muscles, with the exception of slight stiffness of the left arm and leg. The child did not wake from this sopor. Towards evening the temperature was 38.5° (101.2° F.); pulse 130, irregular; fontanelle arched; the eruption bluish; perspiration over the whole body; respiration slow but deep, stertorous, and irregular. Examination of lungs gave evidence of an increase of the catarrh; later, the respiration became more frequent—48, very superficial and irregular; no more vomiting; no passage from the bowels; no stiffness of the neck or back; abdomen not depressed; evening temperature, 38.8° (101.7° F.). (Fourth day.) In the night another long attack of convulsions; morning temperature, 38.7° (101.5°); pulse cannot be counted, very small and irregular; respiration irregular, superficial; occasionally snoring; face pale, conjunctivæ also; the skin over the whole surface of the body somewhat livid, in consequence of the weakness of the heart; fontanelle arched, but without pulsation; pupils oval, of moderate size, without reaction (no ophthalmoscopic examination made); the muscular system relaxed, including the muscles supplied by the facial nerves; involuntary discharges; deglutition cannot be excited; in the afternoon cessation of respiration and of the heart's action.

Autopsy.—Catarrh of the larynx and trachea; capillary bronchitis, with commencing lobular infiltration, and atelectasis of the lower borders of both lungs posteriorly. *Bronchial glands, and all the glands of the body perfectly healthy.* No suppuration in the bones or periosteum. No trace of miliary tubercles anywhere in the body. Stomach, intestines, liver, and kidneys present only unimportant lesions; *spleen not swollen.*

Brain: The two layers of the dura not easily separable; sinus longitudinalis contained a few small, fresh coagula; no marked injection; no alteration of the inner surface of the dura.

Pia friable; vessels filled to the smallest branches, but the single vessels do not contain an unusual quantity of blood; there is no great cloudiness of the tissues, and no pus visible to the naked eye. Pia dry; no change in its external surface (arachnoidea visceralis), and no deposit on its surface that can be scraped off. Surface of brain compressed against the inner surface of the cranium, the gyri flattened and wide, and the sulci small or obliterated.

The outer part of the substance of the brain firm and compact, but its consistency diminished pretty rapidly towards the interior; it contained a moderate amount of blood; few bloody points were apparent, and no rosy tinge of the cut surface. All the ventricles greatly distended. *Plexus chorioideus, in contrast to the pia of the convexity, distended with blood*, and containing a few extravasations as large as the head of a pin; the ependyma unaltered, as far as macroscopic examination could discover, but intact only over the ganglia and the anterior pillars of the fornix, while the fornix, the lower part of the corpus callosum, the psalterium, and the adjacent parts of the brain, were in a condition of great hydrocephalic softening (autopsy twelve hours after death). The quantity of the ventricular fluid was not accurately ascertained; near the floor of the ventricles it was rendered slightly turbid by the débris of the white softening collected there, but otherwise it was clear and not tinged yellow. The aquæductus Sylvii was somewhat dilated. No essential change in the fourth ventricle. Base: The pia somewhat hyperæmic, and apparently more swollen and slightly thicker than normal, but presented no cloudiness from exudation; no exudation in the fossa Sylvii. The optic tracts of both sides somewhat compressed; all the other nerves perfectly normal. Careful examination of all parts of the pia absolutely proved the absence of miliary granulations. Small ampullæ on both optic nerves.

A second case which occurred during dentition, in an equally healthy boy, presented the same pathological changes. The fluid, however, was less abundant, and the white softening less extensive. In other cases a microscopical examination was not made.

Pathological Anatomy.—The cranial bones present different degrees of congestion. The fontanelle is arched; intra vitam, this is usually more marked than after death. We have not met with cases in which the affection has caused much separation of the sutures. There are no changes in the dura. The surface of the brain always presents the signs of great pressure outwards from the ventricles—flattened gyri, disappearance of the sulci.

The pia is usually unchanged to the naked eye: it is apparently not thickened; it contains a moderate quantity of blood, or is anæmic. No traces of arterial hyperæmia can be found, no matter how carefully the arteries are examined. The veins are moderately filled.

A rather more evident hyperæmia is found not unfrequently at the base, particularly near the chiasm, where the blood cannot be so easily pressed out as on the convexity. Even here, there are no macroscopic traces of an infiltration of pus.

We have never found any fluid between the dura and pia (the so-called sac of the arachnoid). The outer surface of the pia is usually remarkably dry.

The cortex and white substance are compressed and but moderately filled with blood. There is no great capillary injection; the whole tissue towards the outer part of the organ looks as if it were pressed out; it is dry and firm. No capillary extravasations are found in the cortex as in tubercular meningitis.

If hydrocephalic softening exist around the ventricles, the substance of the brain becomes more and more succulent from the cortex inwards.

The dilatation of the ventricles is usually symmetrical; when it appears to be unsymmetrical, the appearances are generally deceptive. The existence of a close connection between paralysis and convulsions on one side and a greater distention of the ventricle of the opposite side, is imaginary.

The white softening does not seem to be as extensive as in tubercular meningitis. The results of the microscopic examination compel us to regard it as a *post-mortem* change.

The alterations in the plexus chorioidei are very important. The hyperæmia here is more marked than in the superficial portions of the pia, which are compressed against the bones. The condition may therefore be considered as more like that which exists during life. Their tissue is firmer and more voluminous than normal, and frequently contains punctiform extravasations. The aquæductus Sylvii is sometimes, but not always, dilated; the same is true of the fourth ventricle. The ependyma, as far as it is preserved, does not present the slightest macroscopic change.

No exudation at the base is to be found; the ampullary enlargement on the sheath of the optic nerve, near the bulb, resembles that found in tubercular meningitis. (See article on meningitis tuberculosa.)

Microscopic examination of pia in the above case:

Wandering white blood-cells were found everywhere in the tissue of the pia, but not in such numbers as to present to the naked eye an appearance of milky cloudiness or of purulent infiltration of the membrane. Each small vessel is accompanied by a single, sometimes by a double row of white cells on both sides. Here and there the white cells are collected in small clusters in the lymphatic spaces.

Few are to be found in the parts of the connective tissue that are distant from the vessels. These white blood-cells are entirely unchanged, present no prolongations or changes in shape, and are at most somewhat shrunken. The process is most marked around the chiasma as far back as the posterior border of the pons, and in the fossæ Sylvii; but no trace of pus could be detected by the eye. Behind the anterior border of the pons the process diminished in intensity, but it was still evident in the pia over the medulla oblongata. It was very distinct over the corpora quadrigemina and around the scissura cerebri transversa.

Cortex: The wandering cells were found here also without difficulty, but in less numbers, the process being much milder than in meningitis tuberculosa. In some spots there were small extravasations of red corpuscles, not visible to the naked eye.

In the white substance of the hemispheres wandering cells were not found. It is hardly possible that they could have been overlooked, as they are more easily seen there than in any other parts of the brain. On the other hand, in the superficial layers of the corpora quadrigemina they were again found. Further back in the medulla oblongata nothing was found.

The plexuses were macroscopically swollen, and firmer than normal. Their vessels were greatly distended with blood, and there were extravasations of red corpuscles in great numbers. The connective tissue between the blood-vessels contained many white blood corpuscles, but no characteristic groups of wandering cells were seen. The epithelium of the plexuses was partly separated, and small shreds of it floated about in the fluid in the ventricles.

The ependyma showed no change (hardened specimen); no connective tissue was found between the neuroglia and the epithelial layer. No dilatation of the vessels or migration of cells could be discovered here.

In the spots of white softening no elements, indicating the occurrence of inflammation or necrosis during life, were found. The surface of the macerated spots was fissured, and the epithelium thrown off, but there were no traces of wandering cells.

A number of detached epithelial cells of the ependyma without cilia were found in the fluid at the bottom of the ventricles. A few white blood corpuscles were also found there.

The necessary information in regard to the hydrocephalic fluid will be found in the article on meningitis tuberculosa.

In the majority of cases the remaining pathological appearances are negative or irrelevant. This can be ascertained without difficulty by a consideration of the etiological influences. The lesions do not reveal any physical cause for the process which is in many respects so enigmatical.

These anatomical appearances (which we cannot even say are always to be found) are not sufficient to give a clear idea of the affection. It is noticeable that in the cadaver no trace of arterial hyperæmia is to be found. We are forced to assume that it disappeared at the moment of death as well as in consequence of the pressure of the surface of the brain against the cranium. This seems plausible, when we bear in mind the greater congestion of the plexuses, which are not exposed to such pressure, and the analogous condition of the pia at the base of the brain, the vessels of which cannot, like those of the pia of the convexity, be completely emptied by the pressure. It must be admitted, however, that there is not yet evidence enough to prove with certainty that the process is an inflammatory one, in the ordinary sense of the term. Where was the irritation which, acting on the tissue of the pia, calls forth an inflammatory process in it? We must confess that for the present we are obliged to attribute the abundant transudation and the migration of the cells to the initial increase of tension in the vessels, with perhaps the addition of reflex dilatation of the vessels—the proof of which is, however, still wanting. The question, where hyperæmia ceases, and inflammation begins, cannot be answered by the anatomical evidence. Moreover, a hydrocephalic effusion, arising from venous stasis, can bring about the same changes. Dilatation of the ventricles, and increase of the fluid, and a greater firmness and swelling of the plexuses, accompany this form of effusion also. To distinguish it from the inflammatory form, we have sometimes, but not always, thickening and granulations of the ependyma, which have been wrongly put forward as products of two inflammatory processes; also a milky cloudiness of the pia and serous effusion into its meshes. The cloudiness is due to an infiltration of the tissue with blood corpuscles. There is usually also atrophy of the brain, varying in degree. The question naturally suggests itself, whether these should also be regarded as inflammatory changes. For the effects are the same, only they are produced more slowly; time is allowed for the brain to become adapted to the changes, the adaptation being facilitated, in many cases, by nutritive disturbances of the organ which terminate in atrophy.

If it be true that an altered condition of vascular pressure is able alone to produce abundant transudation and migration of cells in the brain, a second question at once presents itself: Why does this occur more frequently in the brains of children than in those of adults? It is true that a form of meningeal inflammation occurs in adults which resembles very closely, especially in the anatomical appearances, the affection under consideration; but it is distinguished by a much longer, almost chronic course. We are forced to assume that certain special conditions exist in the brains of children, which produce the differences in the results of hyperæmia. Little can be added to what has already been said on the subject.

1. The undoubtedly much greater delicacy and dilatability of the cerebral vessels in childhood are to be remembered.

2. In infancy, in consequence of the expansibility of the cranium, especially of particular parts of it (fontanelles), there is greater room for hyperæmia and transudation.

3. The processes of growth and assimilation are more active in the brain of childhood. An active transudation will occur even under normal conditions, and resorption is also more active. If abnormal conditions, such as increased action of the heart, and dilatation of the vessels be present, it is easy to imagine that an inflammation might be kindled by exciting causes which would not produce the same effect in adults.

It would be profitable to examine this question more closely, but we frankly admit our inability to place the discussion of the question on a firm physiological basis.

Even the mode of production of the effusion into the ventricles is not yet understood. The amount of the normal cerebral pressure is (Leyden and Jolly) from one hundred and nine to one hundred and ten mm. water. The pressure of the tissues and the blood pressure are both included in this; when death takes place, the pressure becomes much less, but it has been proved that it never entirely disappears (Hitzig). The brain is always somewhat compressed during life by the fluid between the dura and the pia (Hitzig). After death it expands from its elasticity, and in so doing the fluid between the dura and the pia is without doubt forced into the brain. We must consequently assume that after death the brain always increases somewhat in size, and that this increase is due to the absorption of fluid from without. The ventricles are therefore somewhat smaller during life than after death, and the small amount of fluid found in them (allowance being made for what is lost during examination) is sufficient to fill them. When the vessels are healthy and not dilated, a certain equilibrium of these forces, which is not necessary for the normal nutrition of the tissues, is everywhere maintained. The most powerful factor in disturbing this equilibrium is an alteration of the blood pressure.

A great disturbance of this equilibrium in the brains of children is, however, demonstrated by the post-mortem appearances of the affection under consideration. Even when the autopsy is made at the earliest possible moment after death, no trace of fluid is found between the dura and the pia, and none in the meshes of the pia. The brain is usually dry and sapless, and is pressed against the cranium by a force acting from within. The ventricles are distended by an abnormal amount of fluid. This fluid can only come from the diseased plexus chorioidei. The rest of the pia are altered in the same way; but there is no fluid effusion on its surface, *i.e.*, no hydrocephalus externus. During life the arching and pulsation of the fontanelle gave evidence of a constantly increasing pressure from within; the diminution in the force of the pulsations of the fontanelle, at a time when the heart's actions were still nearly normal, indicated that the internal pressure had increased to such an extent that it finally presented an obstruction to the entrance of blood into the brain; can we explain the power which forces the serum of the blood into the ventricles?

We are inclined to believe that this is impossible without the supposition that, under the influence of the entire process, some nutritive disturbances, as yet unknown, are undergone by the delicate vessels of the pia, in consequence of which a rapid transudation of fluid and migration of white cells become possible. Whether this is simply coincident with an increase of blood pressure from the action of the left ventricle and a consecutive dilatation of the naturally delicate vessels, it is impossible to say; it must, however, be claimed as a peculiar characteristic of the cerebral vessels during childhood. The influence of fever is also to be considered. The process can then be described as follows:

a. Marked increase of the heart's action sets in, accompanied by a dilatation of the delicate cerebral vessels, particularly in the pia and the plexus chorioidei; a dilatation of the vessels, through the influence of the vaso-motor nerves, may perhaps occur; arterial hyperæmia is present, which will last for a variable time, until the supposed alteration in the texture of the vessels takes place.

b. After this is established, the abnormal transudation and the migration of white cells begin. This may not continue long enough to distend the ventricles and cause pressure upon the brain. The transudation will take place most easily into the ventricles, because a great number of vessels, crowded into a small space (plexus), are to be found there.

c. Every systole will force through the wall of the vessels on the surface of the brain, as well as in the ventricles, a certain amount of fluid, which under normal circumstances would be reabsorbed.

d. Experience shows that this reabsorption does not take place. A cause for this may perhaps be found in the compression of the perivascular lymph spaces by the dilated vessels, and also in the compression of the tissue spaces in all the other absorbing parts (surface of the brain).

e. As long as the pulse wave is impelled into the cerebral vessels, the transudation from the vessels of the pia will continue, particularly from the smallest arteries and the capillaries, in which the pressure is most increased.

f. This of course has a limit; a time comes when the pressure outside the vessels becomes equal to that within them. This is the time when the arterial hyperæmia disappears. The brain, however, is probably not yet in the condition in which we find it after death. It is possible that the process which is described by Hitzig may occur at this time, or even sooner, and that the peripheral fluid may disappear entirely after death.

We have been unable to find any signs of an implication of the ependyma in this process, and are therefore compelled to discard the theory that an isolated inflammation of the ependyma is the cause of the hydrocephalic effusion. Indeed, it is difficult to see *à priori* how the ependyma could take part in a transudation of this sort. Even the influence of the chronic changes in the ependyma on the production of chronic collections of fluid has been greatly overestimated.

The course of the process, as above described, and its symptoms, may be very acute (see the case here reported). But this is not always the case; there are cases which run a very slow course, and still others in which the process presents alternate periods of quiescence and of desultory, rapid increase. Hence there are forms which may almost be called chronic. The duration may be said to vary between five days and three weeks; the cases which run a shorter course being unbrokenly progressive, while the more lingering cases are interrupted by periods of deceptive improvement. Finally, there are brief attacks, lasting only a few hours or one day, which are separated by perfectly normal intervals, and which can only be ascribed to intense cerebral hyperæmia; these naturally suggest the idea that very small exudations may be reabsorbed, until, finally, a fresh and more violent attack proves fatal. The course of the disease is consequently very variable.

It is very important to know *that* (unlike meningitis tuberculosa) *the process may come to a stand-still in any stage of its course.* This, however, is only possible as long as there is no essential obstruction to the cerebral circulation. A few cases are known which recovered after symptoms indicating a large cerebral effusion had existed. In some cases, moreover, a *chronic hydrocephalus* is developed; in fact, the disease under consideration must be regarded as one of the chief exciting causes of the last-mentioned affection. It is uncertain whether this mode of termination is due to influences which keep up a slow, progressive transudation, or to a complete interruption of the process of resorption; at all events, in chronic hydrocephalus very complicated conditions are met with. This possibility of an interruption of the process, and the existence of cases in which recovery really took place, will again be spoken of.

Symptoms.—It is a fact that there is no symptom of this affection which enables us to distinguish it with positiveness from tubercular meningitis. The clinical history may differ in no important particular from that of the tubercular affection.

A comparison of a number of cases of the meningitis of children reveals many differences in the symptoms in different cases.

It follows from the nature of the etiological influences that the prodromal symptoms are not very numerous, and that the duration of this stage is not long; in tubercular meningitis, on the other hand, the reverse is true. Probably the miliary granulations in the pia are developed during this stage; while the circulatory disturbances which form the basis of meningitis infantum begin suddenly (eruptive fevers, fever attendant upon affections of the lungs, upon acute inflammation of the scalp, etc.). It must be remembered, however, that numerous brief attacks of nervous symptoms, such as have been described when speaking of the cerebral hyperæmia of children, have often preceded the final attack, in which the symptoms become threatening.

The fact that certain causes will produce symptoms of cerebral hyperæmia in some children, and not in others, justifies the assumption of a *predisposition*, the nature of which is still entirely unknown.

The mode of invasion of the disease varies. In some cases there is first a slight febrile condition, which can usually be explained by some other affection, but is sometimes inexplicable. This fever is not attended by any characteristic symptoms. In some cases the beginning of the initial fever is marked by an attack of convulsions, which must be classed with the still only imperfectly understood convulsions which usher in severe febrile affections—ex. gr., pneumonia, during childhood. But the attack passes over, and gives no hint of a fatal termination. The febrile condition, however, continues, and soon the symptoms of intense cerebral hyperæmia, which have been already described, set in. Older children complain of violent headache; in younger ones there is restlessness, sleeplessness, strong pulsation of the fontanelles, without arching; in older children there is delirium, or peevishness and moroseness. Vomiting is frequent; constipation is always present. In young children a slight convulsive twitching of the extremities is often observed, though the voluntary movements are in no way impaired; during sleep the eyelids are not entirely closed, the eyes frequently roll about slightly, the pupils are usually somewhat contracted, but react well. The children are very sensitive to light and sound; there is often great hyperæsthesia of the skin, the slightest touch causing pain. The older children are either unable to walk from giddiness, or totter when they attempt to move; they complain of whistling and roaring noises in the ears, are indifferent to everything around them, fretful, and rarely answer when spoken to. The expression of the face is altered; there is a fixed look, or a vexed, angry expression. The countenance frequently becomes distorted, the forehead wrinkled, and a distressing moaning follows. The vomiting is obstinate, even when the diet is carefully regulated. The vomited matters consist of fragments of ingesta, and of gastric mucus mixed with bile; other admixtures are not found at this stage of the affection. The constipation is not so constant as the vomiting, but is usually present. It does not yield readily to a laxative, and soon returns. The fever continues, but varies in degree.

After these symptoms have persisted with varying intensity for perhaps two or three days, the scene may be entirely changed in a moment by the sudden appearance of *general eclamptic convulsions*. The child loses consciousness; the gaze is fixed and entirely expressionless; the eyeballs roll about in every direction at first, then usually remain for a time turned upwards, often in a slightly convergent or divergent position; their direction is altered several times during the convulsions. Then nystagmus-like motions of both eyeballs in their abnormal position occur; convulsive contractions of the muscles supplied by the facial appear, twitching of one or both corners of the mouth, spasmodic and rapidly repeated pointing of the mouth, grimaces and the like, also gnashing of the teeth, and frequently temporary lock-jaw. There is absolute insensibility to external irritation. The convulsion usually travels downwards on one side of the body, involving first the tract of the accessorius, then the upper and then the lower extremity of one side, and finally crosses to the other side, and attacks its muscles from below upwards. It does not always follow this route, however. The muscles of the trunk are affected in very varying degrees. It will be noticed that the spasms of the trunk are of a more tonic character, while those of the extremities are clonic.

Spasm of the glottis is indicated by long-drawn, crowing inspiration and impeded expiration, the immediate occurrence of cyanosis, and powerful contractions of the diaphragm, provided it be free from spasm. Tonic spasm of the inspiratory muscles will retain the thorax in the position of inspiration, so that the normal respiratory movements almost entirely disappear, and respiration is carried on by the diaphragm alone; very frequently it is also seized with spasm and the respiration then stops completely. Clonic spasms of the muscles of respiration lead to rapid and noisy inspiration and expiration. Tonic contraction of the abdominal muscles is not rare, but clonic spasms are. The convulsions vary greatly towards the end of the attack; they cease in the extremities, while they continue in the trunk, or, vice versa, they continue in the extremities, but cease on the trunk, and the regular respiration is then re-established: or they cease entirely, except in the head. Sometimes the attack lasts only five minutes, but it may continue for hours, alternately ceasing and beginning again, and the child may never recover from the convulsions. There is absolute unconsciousness during the entire attack. The pupils vary; frequently they are moderately distended during the attack, fixed, and without reaction; they are often dilated to the maximum extent, and immovable; now and then a momentary rapid dilatation and contraction are observed. The pulse is very rapid, sometimes irregular and intermittent. The previously high temperature sometimes becomes excessive during the convulsion (40° C., 104° F., and over), and afterwards returns to its previous point. This rise of temperature during the convulsion does not, however, seem to be constant.

Death may occur during the first attack. A number of influences combine to bring about this result, the most important of which is the disturbance of respiration. The interference with and suspension of the respiratory movements, caused by the tonic spasms, may produce such a condition of the blood, that the excitability of the respiratory centre is lost; consequently the normal respiratory movements are not re-established on the cessation of the convulsions. An attack of this kind is therefore made up of the symptoms of a violent cerebral hyperæmia and a terminal convulsion, which proves fatal by paralysis of respiration; none of the signs of internal pressure are developed. It is rare, however, for the attack to follow this course.

If the eclamptic attack does not prove fatal, the further course of the affection is marked chiefly by an aggravation of the cephalic symptoms, in which there are frequently temporary remissions and improvements. These are, however, delusive in the majority of the cases.

The disturbance of the intellectual functions increases rapidly; there is great listlessness and indifference; small children lie in an unquiet sleep, with tightly closed lids, frequently groaning and giving signs of pain; older patients lie for hours at a time in a state of stupor.

This is the time when the hydrocephalic cry (Coindet) is frequent; it is not, however, characteristic of the meningeal affection, as it is heard often in typhoid fever and in other affections. The functions of the organs of sense are already disturbed, or at least no reaction is produced by the application of adequate irritation. The children do not fix the eyes upon any object; they appear, in many cases, to see only very indistinctly, or not at all. There is pulsation of the fontanelle in small children, which, however, becomes weaker as the pressure from within increases; the fontanelle gradually arches outwards, and is often very sensitive to pressure. The pupils are now usually moderately dilated, and lose their normal reaction, while in the early part of the disease they were narrow, and reacted normally. The reaction at first becomes slow, and may entirely disappear in a day. The pupils are frequently irregular in shape. Great dilatation of both pupils, or dilatation of only one of them (partial oculo-motor paralysis), we have never seen. The pulse begins to fall; the stage of depression of the pulse is often of short duration and often appears to be absent, because the pulse is not examined while the child is perfectly quiet. Every agitation, every movement of the child, will cause a rapid increase in its frequency. At one time the pulse may be 60–70, and an hour later it may be 160–180; it is usually irregular, though this is not so apparent when it is slow; it is then usually tolerably full, but it is small and weak when rapid. The irregularity is due, in some cases, not to an irregularity in the rhythm of the heart's action, but to inequalities in the force of the contractions, in consequence of which a pulse-wave is occasionally lost in the arteries. The respiration also becomes irregular. This should cause alarm in older children; in infants it is naturally irregular. The fever continues, but is exceedingly irregular; no rule can be formulated for it, because the initial fever, which is often dependent on other lesions, complicates that caused by the cerebral process. Even when uncomplicated, however, its curve is found to be exceedingly irregular. There is usually fever, morning and evening: remission in the morning, exacerbation in the evening—variations which are often nothing more than the normal daily variations. Frequently an excessively high temperature, for which no cause can be discovered, is observed at noon, during the afternoon, or in the middle of the night. Often also an irregular fall in the temperature occurs; towards the end of protracted cases the curve shows peculiarities, which will be described further on.

Eclamptic attacks may terminate the affection; the children frequently die, after a long series of irregular convulsions, which do not belong to any particular type, but attack at one time the muscles of the head, at another time those of respiration, and again those of the extremities; or convulsive attacks of milder grade and of less importance may occur: gnashing of the teeth, rolling of the eyes, chewing or sucking, or whistling movements, which are continued for hours, winking of the eyelids. Stiffness of the muscles of the neck and back is usually, but not always, observed at this time; turning the head causes a cry of pain. This symptom in some cases will be observed, even in an earlier stage; it is frequently transitory and discontinuous. Convulsive twitchings of the extremities are quite common; the voluntary movements become less and less frequent, and those which are still made with the extremities, though evidently made with a purpose, have the appearances of entirely unconscious, reflex acts. All this time vomiting continues; the vomited matter rarely contains more bile than at an earlier period of the disease, but is composed of a slimy fluid, containing flocculi and an immense number of bacteria and of other indeterminable small organisms of a vegetable nature ; it is customary to infer from these appearances a commencing softening of the stomach. Blood is, in rare cases, found in the vomited matter (Bednar). In these cases small hemorrhagic infarctions of the mucous membrane of the stomach are discovered. The boat-shaped retraction of the abdomen occurs in this affection, as well as in meningitis tuberculosa, but seems to be less constant ; we have found it to be absent in several cases. The constipation is obstinate. Occasionally the ability to swallow is lost, sometimes at a very early stage of the disease.

The nutrition and appearance of the children are by this time much altered ; stout hearty children rapidly become emaciated ; the skin is mostly dry, perspiration occurring principally on the head. Transitory, circumscribed hyperæmias are very apt to follow slight injuries or blows or pressure with the finger. It appears to be characteristic of this affection that paralyses of the face (strabismus convergens and divergens, ptosis, facial paralysis) and of the extremities are comparatively very rarely observed ; they certainly do occur, but much less frequently than in meningitis tuberculosa, in which basal exudation is often so abundant.

The patient gradually sinks into a condition of deep coma, which often lasts for a very long time. Intercurrent awakening, lucid intervals, and the like, we have never seen. During this stage, and sometimes even earlier, contractions of the extremities occur ; there is often a tetanic spasm of the muscles of the neck and extremities, so that the child can be lifted like a cadaver in which rigor mortis has set in. It is even possible that convulsions of different kinds may take place ; but contractions are the prevailing motor symptoms of this last stage. Of a preservation of the senses, there is, of course, no longer any question ; the reflex excitability, even that of the spinal cord, disappears slowly but completely, although occasionally, during the course of the disease, an increased excitability has existed. The position of the eyeballs varies greatly, and no law can be formulated for it ; there is sometimes a complete relaxation of the whole muscular apparatus of the eye. The very rapid pulse gradually becomes more irregular, and is often marked by relatively long intermissions. The action of the heart is now really irregular; the respiration is also exceedingly irregular, presents long intermissions, and finally ceases, after an agony which is often very protracted.

The fever during the last stage varies; it is often very irregular, apparently following no fixed law. Near death the temperature often falls below the normal. On the other hand, there are cases, which are usually more rapidly fatal, in which the fever increases up to the time of death, and even a hyperpyretic temperature may be reached.

Urine: The results of but few thorough examinations have been recorded, but they, as well as those made by myself, show that no characteristic sign of the disease can be discovered in the urine. It is concentrated, throws down a copious uric acid sediment, and contains a greatly increased quantity of urates; the sulphates are said to be increased, the phosphates to be diminished (this we can confirm); the chlorides are said to be somewhat increased (Bednar); we, however, have found them diminished. In one case there was a marked increase of indican.

Variations in the course of the disease.—All cases are not fatal. There is reason to believe that some of the reported cases of recovery from tubercular meningitis, which are scattered through the literature of the subject, really belong in this category.

a. A remission of the symptoms may occur even when the existence of a ventricular effusion is beyond doubt. The symptoms slowly diminish in intensity, the fever disappears, the cerebral functions are re-established, and, in a few rare cases, complete restitutio ad integrum seems to take place. We have ourselves never met with such a case, though we have seen several cases in which partial recovery took place. One child recovered with impaired intellect; no enlargement of the cranium could be observed. In a second case the character was altered; even during boyhood the patient performed all sorts of perverse actions, his inclinations were perverted, and he was subject to feelings of intense anxiety (there was in this case, however, marked hereditary taint). The resulting condition of the brain may be of a complex nature; it is usually, and probably correctly, classed with acquired chronic hydrocephalus—although this term indicates the nature of the change as little as "hydrocephalus acutus" indicates that of the original affection, since atrophy of the brain, especially of its cortical portion, takes an important part in the change.

b. The symptoms before death may be very much prolonged. In some cases the affection lasts thirty days or even longer, reckoning from the appearance of the first symptom. In these cases the symptoms are developed very slowly, the fever is very irregular, and on some days is entirely absent; remissions of the cephalic symptoms occur, which are soon followed by aggravations. The progressive aggravation of the condition, from day to day, which marks the ordinary cases, is not observed. The increase of the intracerebral pressure is slow, but finally leads to a fatal termination.

c. Cases of moderate duration (from nine to fourteen days) tally pretty closely with the description already given.

d. Finally, there are cases which run a very rapid course, and terminate with eclamptic convulsions, before the unquestionable signs of a ventricular effusion have presented themselves. Such cases we have had no opportunity of examining anatomically.

Prognosis.—This is very unfavorable, though less so than in meningitis tuberculosa. It is impossible to say what the percentage of recoveries is; the experience of a single individual is of no value. There is uncertainty also with regard to the sequelæ. In one case, the child after recovery from the hydrocephalus was afflicted for a long time with great deficiency of intellect, but suddenly a rapid mental development began, and eventually a high degree of intellectual capacity was attained. As long as our knowledge of the possibilities of reabsorption and of the secondary alterations in the cortex is so limited, nothing can be said with regard to these chronic processes which will be of any permanent value.

The diagnosis will be spoken of later in connection with that of meningitis tuberculosa. As the therapeutics also, of this affection and of tubercular meningitis, are precisely similar, we will defer the discussion of them to the conclusion of the article on the latter affection.

II.—*Meningitis Tuberculosa.*

Meningitis Basalis. Hydrocephalus Acutus.

Tuberculous meningitis resembles closely the affection of childhood, treated of in the previous article. The anatomical appearances are in part the same. The symptoms and course are often so similar, that it is impossible to say which form of the meningeal affection we have before us. Tubercular meningitis, however, is signalized by the fact that its etiology is evident, and the existence in this case, of a definite exciting cause, naturally suggests the idea that the meningitis of childhood may be due to possibly similar, but as yet entirely unknown causes. We have already stated that our knowledge is not yet sufficient to enable us to explain the etiology in a satisfactory manner.

The specific irritation of the pia which leads to tubercular meningitis is the development in it of miliary tubercles. It was a long time before these were discovered, and before it was possible to separate, upon the basis of the anatomical lesions, meningitis tuberculosa from the other affections of the pia that also lead to effusions into the ventricles. After the discovery, by Coindet and Papavoine, of miliary granulations, observers fell into the opposite mistake, and attributed every case to miliary tuberculosis of the pia, until the more precise methods of examination, employed in recent times, threw a clearer, though still insufficient light upon the subject. The development of these miliary granulations in the pia and brain is subject to very great variations; sometimes they are numerous, sometimes few in number; in one case the whole pia may be filled with them, and in another only part of the membrane is affected; they may all be developed together, or we may find them in different stages of development. The pia tolerates their presence for a period which varies of course in different cases, but finally a reaction is excited, which terminates in inflammation. This inflammation in the majority of cases is very intense, so much so that extensive macroscopic collections of pus are formed in a very short time; at the same time a ventricular effusion takes place, to which the remarks made in the last chapter will also apply.

The fact that the invariable exciting cause of the disease is the presence of a definite, specific form element, raises the question as to the origin of this elementary substance. It is consequently necessary to cast a glance at the composition and origin of miliary tubercles.

The Elements of Miliary Tubercles.

Virchow has shown that what are usually called miliary tubercles, are composed of a number of submiliary nodules, and that, consequently, the size of the masses is not characteristic. Even the smallest miliary tubercles are made up of still smaller elements, as Rindfleisch has demonstrated in the tubercles of the lungs. The formation of very large nodules (of the size of peas, or beans) is no rarity, but is not the rule. The agglomeration usually attains the size of the bodies to which the name of miliary tubercles is ordinarily given. The elements of the miliary granulations are not always the same. Differences in structure have been pointed out especially by Rindfleisch, who sharply differentiates the fibroid structure of certain tubercles, from the small-cell texture of the majority of the miliary granulations, and from the lymph-adenoid nature of the tubercles of scrofula. These distinctions may be of great importance, but they are not yet clearly understood, and for the present are clinically of no value.

The miliary tubercles of the pia are composed of the following elements:

a. Of rounded or irregularly rounded masses of protoplasm consisting of a dense, strongly refracting substance, with sharp contours, and without investing membranes. Each of these cells contains a round, moderately brilliant nucleus, which is often situated near the surface. The cells vary greatly in size. We frequently find some that contain two or three nuclei, and constrictions of a few nuclei indicate their mode of production.

b. In addition to these elements, which are under all circumstances very numerous, giant cells are also found. These are probably more an accessory than an essential element. They are simply large irregularly formed masses of protoplasm, entirely similar in structure to the above-mentioned cells. They contain a great number of nuclei, some of which resemble the nuclei of the smaller cells, while others appear to be undergoing a higher development to new cell forms (Rindfleisch). The position of these giant cells is not constant; they are found within and on the periphery of the tubercles, and Rindfleisch (certainly good authority) does not seem to look on them as specially important elements of the tubercle.

c. There are also very small and numerous cell elements, derived from the masses of protoplasm described under *a.*, by endogenous cell formation. The nuclei are the same, but the protoplasm is much less abundant, and also less dense and less strongly refracting than the mother-cells.

d. Finally, the tubercle possesses a fibrous stroma, which in many places resembles the delicate stroma of a lymph-follicle; it is made up of rounded and exceedingly delicate threads of protoplasm, which are connected with one another in every direction.

Origin of the Miliary Tubercle.

The mother soil of the miliary tubercle is the endothelial lining of the vascular and lymphatic system of the human frame. The miliary tubercle is produced by the action of the specific irritation upon the endothelial cells of the lymph vessels, the serous membranes, and the smallest blood-vessels. We have found (although we do not claim to be an authority on histology) that tubercles are developed not only on the outside, but also on the inside of the blood-vessels, and that they are able to perforate the walls of the vessels from within outwards; a perforation from without inwards is universally admitted to take place frequently. The tubercles are developed in the course of the blood-vessels, because the vessels are usually, and in the brain and its membranes always, accompanied by lymphatic canals, which are lined with endothelium, and because the vessels themselves possess an endothelial lining. When one of those scale-like endothelial formations, which are situated upon the outer side of the adventitia of the small vessels of the pia and cerebrum, is subjected to the specific irritation, the development of the miliary tubercle begins. The endothelial scale contains a nucleus and a small amount of protoplasmatic substance, which can be demonstrated without special preparation, but does not possess a sharp margin. According to His, the margin can be made visible by staining with nitrate of silver. The first change is an increase of the protoplasmatic substance; then the nucleus divides, and the fragments become more globular, so that they already resemble the previously described nuclei of the tubercle cell. The whole may now resemble a giant cell, but usually fissures quickly appear in the protoplasm, and a number of small cells, with single nuclei and without membranes, are formed. These are the above-mentioned prevailing elements of tubercle.

Secondary Changes.

The tubercle macroscopically assumes a yellowish-white color, which appears first in the centre and spreads towards the periphery. The microscope shows this to be due to the occurrence of a granular and fatty degeneration. The final result of this process is the change of the cellular elements into a mass of small elements, in which the cellular structure can no longer be recognized, and amongst which great quantities of formless detritus are found. This is, without doubt, to be attributed to a dry necrosis, due to the absence of vessels in the growth. It results, according to Rindfleisch, from the interruption in the continuity of the very small vessels, in the wall of which the tubercle has been formed. The vessel is entirely obliterated by the new-growth. This dry, caseous degeneration is modified in character by the inflammatory reaction which the tubercle excites in the surrounding vascular tissue, for the tubercle acts as an intense irritant to the tissue on which it is seated. The primary tubercle, with its modifications, is, therefore, to be distinguished from the secondary inflammatory products and the changes they undergo. In the pia this is not so difficult, and not so important as elsewhere, because secondary changes of the inflammatory products occur only in a very small minority of the cases. The life of the patient is destroyed before they can occur.

Relation of the Miliary Tubercle to Caseous Collections in the Body.

It is certain that a definite connection exists between caseous inflammatory products anywhere in the body and the miliary tubercle. The introduction of caseous detritus into the circulation of an individual causes the development of miliary tubercles. The most minute portion of caseous detritus must be looked upon as an infecting agent, as a poison, which by direct contact with the endothelial structures, incites them to the production of miliary tubercles. The introduction of the poison into the canal system of the body takes place from within, whenever a cheesy focus exists. The mode of infection is not yet known with certainty; it may probably occur in a number of ways.

The manner in which the infection of the pia is brought about—will be discussed later.

The *pathological anatomy* of the affection varies—the appearances in the brain are far from uniform, and, on the other hand, evidences of manifold pre-existing disturbances are found in other parts of the body.

I. *Brain.*

Various changes in the cranial bones may be found, but they have no significance with regard to the affection itself. Caries of the petrous portion of the temporal bone may possess a certain degree of importance, as we will see below.

Tubercles are found in the dura occasionally; they would undoubtedly be found more frequently if the examinations were made more carefully. They do not appear to be situated always in the same layer of the dura; sometimes they are found between the two layers close to small branches of the arteria meningea media, while others appear to be situated in the inner lamella of the dura; in the latter case their relations to the epithelium of this layer are not yet determined. Other anomalies of the dura are very rare: a case of old pachymeningitis without secondary hemorrhage in a phthisical drunkard deserves mention.

The lesions of the pia are as follows:

a. It is dotted with miliary tubercles, which are found without difficulty when the pia is carefully stripped from the brain, and its inner surface examined. They appear as grayish-white nodules with a partly gelatinous appearance, always situated in the immediate neighborhood of vessels; they are very variable in size, some being scarcely visible to the naked eye, while others are as large as a poppy- or a millet-seed, and finally masses, as large as a pea, are found, which have been produced by the confluence of smaller nodules.

The distribution varies greatly; but it must be particularly borne in mind that the tubercles are always to be found close to the vessels. In some cases the whole length of an artery, from its origin in the circle of Willis, is covered with numerous tubercles, while in other, rare cases, the granulations are chiefly seated on the peripheral branches in the superficial parts of the pia.

In this way the convexity may be richly covered with tubercles, while the base has but few; the reverse, however, may be the case. Frequently all the arteries given off from the circle of Willis are dotted with miliary tubercles, so that there is a general filling of the pia with tubercles, although some parts of it contain more than others. Often the tubercles are limited to particular portions of the surface of the brain, such as both frontal and both parietal regions; when the arteries are traced out, it is not difficult to see that the territory supplied by the arteries of the fossa Sylvii and corpus callosum on both sides are affected. Or the new-growths may be most abundant in the pia covering the upper and under surfaces of the cerebellum, or on the median surfaces of the hemispheres, or on the posterior lobes of the brain. In such cases the location of the tubercle corresponds more to the areas of distribution of the arteriæ profundæ and cerebellares. There are cases in which asymmetrical tracts are affected on the two sides, as, for instance, the parts supplied by the arteries of the fossa Sylvii[1] and corpus callosum on one side, and by the artery of the corpus callosum alone on the other side. The tubercles may even be limited to the branches of one artery, as was shown in a case reported by Louis. Two cases came under our own observation in which the tubercles were limited to the branches of one artery; in one the territory of the right, in the other that of the left arteria fossæ Sylvii was affected. In both cases the symptoms were perfectly well-marked. These cases will be described presently.

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[1] Vide *Seitz*, Miliary Tuberculosis of the Pia, p. 169.

The number of the tubercles certainly varies as much as their distribution. A limited number may be present and a careful search be necessary to find them, while in other cases they are numbered by thousands. It is noticeable that in some cases an intense inflammation of the pia is excited by a very small number of tubercles, while in other cases the inflammation is not excited until a very large number of granulations have been developed. The grouping follows no fixed law. Many granulations are isolated, and separated from each other by wide intervals, while others are collected together in dense groups. This agglomeration may go on until the granulations are completely confluent, and form, by combining with the inflammatory products of the pia, voluminous thick plaques, which exert a mechanical pressure on the brain.

Finally, the granulations may be found in all stages of development: in some cases indeed all the tubercles are almost similar in color, size, and consistency, but we quite as frequently meet with cases in which there is a confused mingling of the smallest and the medium-sized tubercles, some of which are undergoing granular and fatty degeneration, while others are already completely degenerated. From these differences an essential dissimilarity in the *age* of the tubercles may be inferred, or, in other words, *it may be supposed that in the first class of cases the exciting cause was intense and produced its action at once, while in the second class it was less intense and was frequently repeated during the course of the disease.*

In addition to the tubercles of fresh formation, the traces of older processes are occasionally found in the pia. Old callosities, composed mainly of connective tissue and enclosing caseous miliary tubercles of ancient date, have been found on various parts of the surface of the brain. Such callosities may even undergo a secondary, complete caseous degeneration, and a large superficial cheesy mass, whose origin dates back a long time, may then be found, in the neighborhood of which a new eruption of miliary tubercles may be developed.

Are we able, from the appearance of a miliary tubercle in the pia, to determine its age with accuracy? This is at present impossible, because in all probability the caseous degeneration is more rapid in some localities than in others, and presumably is also more rapid in some individuals than in others. We have as yet been unable to formulate any rules for determining the age of the tubercle, because the beginning of its growth in the pia is not indicated by any reliable symptoms, and hence no statistics could be obtained for comparison.

Are we able to explain why in one case a large number of tubercles has caused no macroscopic suppuration, while in another case the reverse is true? This question too we are unable to answer. Probably the difference is due to some unknown conditions, such as individual peculiarities. Can miliary tubercles exist without causing that form of general purulent inflammation of the pia which, as we will soon see, is the second great lesion of the disease? Experience teaches that the reaction of the pia is usually very acute; the other form of reaction resulting in the formation of connective tissue, callosities, and the encapsulation of tubercles, is rare. This newly formed connective tissue, moreover, is not free from danger; for chronic inflammation of the pia of this sort is seldom entirely arrested, and there is always danger that the caseous degeneration will progress further and that lesions of the surface of the brain will be produced.

B. *The pia shows inflammatory changes.*—These may be both macroscopic and microscopic, or the pia may be found in the same condition as in the meningitis of childhood—that is, without any change visible to the naked eye. This is more apt to be the case with the pia of the convexity; the lesions at the base of the brain are more constant, and we rarely fail to find macroscopic suppuration there. The suppuration is characterized by yellowish cloudy patches in the pia, particularly along the vessels, and by marked swelling of the whole tissue.

The amount of blood contained in the vessels of the pia varies greatly, according to the degree of pressure exerted from the interior of the brain. As this pressure is great in the majority of the cases, a high degree of hyperæmia is the exception; a moderate hyperæmia, however, is frequently present. When the effusion into the ventricle is very abundant, the surface of the pia is dry, and the membrane is thickened and cloudy, to an extent that depends upon the grade of the inflammation at the time the sudden hydrocephalus occurred. The pia may be perfectly transparent, and yet contain a number of miliary tubercles. When there is room, a serous or sero-purulent effusion in the pia is found, but this is wanting in the majority of the cases.

In other cases, which resist the disease somewhat longer, the signs of suppuration on the convexity are more evident; the streaks of pus that accompany the vessels are numerous; the pia is everywhere filled with a sero-fibrinous, yellowish exudation; it has lost its delicacy and transparency; and is infiltrated, inelastic, and easily torn. The lesions at the base are more constant, and, in the majority of the cases in which the signs of inflammation are not distinct on the convexity, they are sufficiently manifest on the base (meningitis basilaris).

The greatly thickened pia around the chiasm, over the infundibular region, the corpora candicantia, and the substantia perforata anterior, and from there into the fissures of Sylvius, and also over the pedunculi and in the angles between them and the gyri uncinati, is filled with a sero-fibrinous yellowish saline exudation. This exudation contains a considerable quantity of pus (migrated white blood corpuscles), which is deposited, particularly along the vessels, in the form of bands. The miliary tubercles are scattered throughout it in greater or less numbers, and in various stages of the granular and fatty degeneration, so that it is often difficult to distinguish the very caseous agglomerations from thickened pus. The tubercular masses, when the caseous degeneration is far advanced, readily crumble under pressure. The changes spread, but usually diminishing in intensity, from the above-mentioned places along the vessels to the surface of the island of Reil, from there over the operculum along the branches ascending from the fossa Sylvii to the convexity; frequently, however, suppuration, visible to the naked eye, is not seen beyond the fossa Sylvii. The infiltration of the pia often extends backwards over the anterior surface of the pons, involves the portion of the pia that covers the medulla oblongata, particularly in the neighborhood of the facialis and acusticus, creeps upwards to the upper surface of the medulla, and implicates the pia of the entire cerebellum, particularly on its under surface and that part of its upper surface which is near the corpora quadrigemina; here, too, the miliary tubercles are found in varying quantities. Finally, from the chiasma the suppurative process of the pia extends forwards to the under and middle surfaces of the anterior lobe along the arteria corporis callosi, and the lobus olfactorius is often found entirely imbedded in a mixed sero-fibrinous and purulent exudation.

These changes are not always symmetrical; if, for example, the tubercles are situated only in the tract of one arteria fossæ Sylvii (see below), the exudation is then found only on one side of the chiasm in the Sylvian fissure; the other side is not entirely normal, but the change is very much less marked. We have also met with cases in which the exudation around the chiasm was very slight, while about the pons and its posterior border upon the under surface of the cerebellum, and from there upwards to the neighborhood of the corpora quadrigemina, it was very abundant. Very few tubercles were found in the parts supplied by the anterior cerebral arteries (fossæ Sylvii, corporis callosi), but they were found in great numbers on the branches of the cerebellar and posterior cerebral arteries. Finally, more or less extensive extravasations of blood are frequently met with in the altered pia of the base.

It is important to bear in mind how favorable the conditions now are for the extension of the inflammation from the base to the plexus chorioidei. The inflammatory process is carried into the descending cornua, by the processes of the pia which enter them on both sides. The inflammation is also transmitted directly from the region of the corpora quadrigemina through the scissura transversa cerebri to the plexus. The affection is so intense, that a yellowish purulent exudation is sometimes found in it.

In general it can be said that the greatest suppuration and exudation will be found where the tubercles are most numerous; there are, however, many exceptions to this, which it is difficult to explain at present.

The extravasations of blood in the pia are found not only at the base, but also on the convexity; in size they vary from a miliary speck to the area of several square inches. The blood is extravasted into the meshes of the pia; we once saw in connection with a rupture of the pia and a superficial lesion of the brain a pool of blood of considerable thickness. It is not difficult to account for this hemorrhage; many authors (Rindfleisch, et al.) have pointed out that the lumen of the vessel is obstructed by the growth of the tubercle in its wall; the media and intima are pressed inwards, so that the passage is narrowed; finally, both media and intima are perforated, as Rindfleisch has shown to be the case also with the small arterial branches in the lungs. Consequently the possibility of hemorrhage is always imminent. Small arterial and venous thromboses also occur in the pia, for the same reasons. If these processes occur in many vessels at the same time, marked disturbances in the circulation of the pia may unquestionably occur. If they occur only in a few places they may be of little importance, in consequence of the numerous anastomotic connections of the vessels of the pia. The venous thrombi may extend into the sinus longitudinalis.

C. *The ventricles are dilated, and contain a hydrocephalic effusion.*

The latter is wanting in a number of cases.

Out of forty-three recorded cases Seitz found ventricular effusion absent in twenty-five per cent. From our own experience we are inclined to believe that this percentage is too large; we think the correct percentage is from eighteen to twenty. This point cannot be settled until we have the records of a greater number of cases at our command.

The effusions present the greatest differences. Small effusions may easily be overlooked in consequence of the imperfections in our methods of examination, while the largest are so excessive as to be alone sufficient to annihilate the functional power of the brain. Even when no effusion of any significance has existed, we have always found the descending cornu dilated —a fact which can be easily explained by the immediate contiguity of the pia of the base. We find not only the lateral and middle ventricles greatly dilated, but often also the aquæductus Sylvii and the fourth ventricle. Occasionally one ventricle is said to have been much more dilated than the others; when we have ourselves met with this appearance, we have attributed it to an error of observation due to bad methods of examination. In the cases in which the tubercles were situated only in one fossa Sylvii, the dilatation was found to be equally great in both ventricles. The character of the fluid is not always the same; it is often purely serous with a slight cloudiness at the base of the ventricle, and sometimes quite cloudy from the admixture of epithelium and white blood corpuscles; purulent effusions are also said to have been observed. The suspended elements are of different kinds; in addition to what have been already mentioned, we find a good deal of débris from the hydrocephalic softening. Sometimes the fluid is slightly tinged with blood, and the source of the bleeding will be found in the plexus chorioidei.

Whether an acute inflammation of the ependyma takes place or not, is still an open question; we have never been able to demonstrate it, although we have often found the vessels under the ependyma distended with blood. It is stated that miliary tubercles have been found on the ependyma; and since we have found them in the tissue of the brain itself, we are more disposed to admit the correctness of the statement. We have never met with a thickening, such as is seen in chronic hydrocephalus.

Great changes are to be found in the plexus, particularly a marked degree of hyperæmia, so that there seems to be an increase in its volume; moreover, miliary tubercles are not unfrequently found in the plexus itself, although never in large numbers, and by no means constantly. A migration of white blood cells, however, similar to the migration that takes place in the hydrocephalus of children, is constantly observed. Finally, small extravasations of blood are frequently found, which are produced in the same way as those in the pia of the convexity.

Hydrocephalic white softening is not a necessary complication: it may be wanting, and, when present, it may present every possible grade. It may involve only a portion of the fornix and the corpus callosum, or it may extend upwards into the contiguous brain tissue, especially in the angle where the corpus callosum is connected with the outer surface of the cerebral ganglia; in a few cases large portions of the ganglia themselves were affected. No connection can be traced between the softening and the amount of the effused liquid. The former is a phenomenon which certainly exerts no influence on the symptoms during life, but which perhaps begins before death, as it has been seen even in the freshest cadavera. We may perhaps assume that the portion of the brain which is especially exposed to pressure, may die even before the death of the patient, and then undergo a rapid maceration.

It follows from the above that these three principal lesions may be combined in different ways, in which the following leading forms—examples of which occur in the practice of every physician—may be distinguished:

I. Miliary tubercles in the entire pia, but usually in small quantities; no pus visible to the naked eye on either base or convexity. No hydrocephalus of any amount; wandering cells can be found in small numbers in the pia by means of the microscope. It is possible that cases may come under examination before the migration of the white blood corpuscles has taken place; any one who desires so to do may then term this miliary tuberculosis of the pia, and regard it as a special form of the disease, but from a clinical point of view it would be incorrect. This form is rare; usually other changes exist, which have caused death.

II. Miliary tubercles in the entire pia; no macroscopic suppuration at the base or on the convexity, but changes in the plexus and hydrocephalus; wandering cells everywhere in the pia. This form also is rare.

III. Miliary tubercles everywhere in the pia; exudation and suppuration at the base, but not extending to the convexity; hydrocephalic effusion. A common form.

IV. The same as in III., with the addition of exudation and suppuration on the convexity. Less frequent than the last.

It is easy to see that these various forms constitute a series, the members of which are distinguished from one another by the intensity of the manifestations of the inflammation.

V. Partial miliary tuberculosis of the pia (territories of one, two, or three arteries); different grades of exudation and suppuration at the base and in the parts supplied by the branches of the affected arteries; wandering cells found usually over a much wider extent of the pia; hydrocephalic effusion varying in quantity, but usually moderate.

VI. Miliary tuberculosis, varying in extent, and accompanied by an old affection of the pia, with circumscribed thickening and laminæ of indurated or caseous connective tissue. Imbedded in these are old miliary tubercles that had long before undergone caseous degeneration. Such cases consequently present the lesions of two tubercular affections of the pia of different ages.

The brain itself undergoes many important changes.

The great anæmia of the cortex and neighboring white substance, and the dry condition found in many cases, are probably the result of pressure, and are almost entirely wanting, if there is no hydrocephalus present. In the latter case there is injection of the pia and of the cortex and neighboring cerebral tissue, together with œdema. This œdema is probably not entirely the result of the death agony, as in addition to it certain inflammatory changes are to be seen; and, from the condition here observed, one may infer what processes had taken place in the brain before the increasing pressure within crowded away the fluid on the surface.

It is difficult to say what becomes of this fluid; probably it is pushed into the brain itself, and we would in that event have in hydrocephalus not merely the fluid coming from the inflamed plexus.

The many small punctiform apoplexies observed in the cortex of the brain produce their injurious effects, as we believe, in a simply mechanical manner. Although cerebral tubercles occur in the cortex, we do not consider the formation of tubercles in the walls of the vessels to be the cause of the hemorrhages in every case. Rindfleisch goes a step further, and considers the tuberculous degeneration of the vasa propria of the brain as the cause of the many ruptures that occur in the walls of the vessels; at any rate, the capillary apoplexies, which may also occur in many other spots (thalamus opticus, corpora quadrigemina, pedunculi, pons, cerebellum, hippocampus major, corpus callosum), act as a mechanical injury to the nerve substance. Exceptionally they may attain a considerable size. Extensive foci of red softening, occasioned by the inflammatory disturbances of the cortex, together with extensive ruptures of the vessels, have been found by us several times in the gyrus uncinatus and fossa Sylvii, and once on the surface of the brain; in fact, they were to be interpreted as the results of a true *tuberculous encephalitis.*

Among the disturbances which occur in the cortex, and which are analogous to those which occur in the pia, we may mention:

a. The development of miliary tubercles in the cortex; they do not seem to be always present, and can hardly be seen with the naked eye; yet in some cases, where they are present in great numbers, they may be distinguished without any great difficulty, after the ordinary preparation of the cortex. A spindle-shaped substance is seen surrounding the small vessel, and obliterating the perivascular lymph space; it may even displace the neighboring brain tissue. On examination this substance will be found to consist of the elements of miliary tubercle. In one case whole rows of such spindle-shaped bodies, one behind another, were found in the cortex of the fossa Sylvii, and extending even into the white substance of the brain. The effect produced by such bodies is partly mechanical, partly the dynamic effect already explained. A great number of tubercles will naturally cause a marked interference with the cerebral elements of the part affected; if hemorrhage is added, the situation becomes worse; the dynamic effect also causes an inflammatory reaction.

b. The escape of elements of the blood in the cortex, indicating the existence of an inflammatory process. In the edge of the ependyma of the cortex, wandering white cells, somewhat shrunken, are found; these perhaps have forced their way in from the pia. Much more important is the migration which takes place from the vessels passing through the cortex, and the products of which are even to be found in the white substance of the hemispheres. Colorless blood corpuscles may be found everywhere in the walls of the vessels, and filling the perivascular lymph spaces; they are crowded in masses in the neighboring glia tissue, but decrease in numbers as we go further away from the vessels, thus leaving no doubt as to the original source from which they came. If the pia be stripped off from the brain, small portions of brain substance will be found clinging to the vessels which pass from it down into the cortex, and the surface of the brain will appear rough—evidently a result of the close connection of the vessels with the surrounding glia tissue. The increased succulence of the white substance and of the cortex can be explained on the supposition of a transudation of elements of the blood in solution from the vessels. Scattered groups of a few red corpuscles make it probable that they also pass through the walls of the vessels. Apoplexies cause large collections of red corpuscles with destruction of the tissue of the glia.

We have never been able to discover any changes in the ganglion cells.

It would be out of place to discuss what becomes of the escaped blood elements, as life ceases before there is an opportunity for further changes to take place. The varied symptoms of impaired intelligence developed in the course of the disease are certainly dependent in an important degree upon the processes here under consideration. They occur in different parts of the brain, but accurate observations are wanting; we have observed them, for instance, on the upper layer of the corpora quadrigemina, and have verified the existence in this locality of miliary tubercles as well as of wandering cells.

We have no personal knowledge regarding the changes in the ependyma of the ventricle; miliary tubercles are found there, and perhaps inflammatory products, but the question is still an open one. Colberg states[1] that in a case of hydrocephalus acutus ventriculorum without basal meningitis, he found neither tubercles nor pus in the pia, but a number of small-celled elements in the ependyma and proliferation of the nuclei of the capillaries. Colberg considered this as a commencing tuberculosis of the ependyma. Such appearances show how much is as yet unknown in regard to the subject.

The cerebral nerves.—As all the roots of the cerebral nerves, under certain circumstances, lie exposed to basal exudation, they all may be more or less affected by it. The injurious influence exerted is not so much that of mechanical pressure, as that of an inflammation spreading to the connective-tissue envelope of the nerves. But that an increase of the intracranial pressure may eventually exert an injurious effect upon the basal nerves, there can be no doubt; it is difficult, however, to discriminate accurately between the different influences.

In many cases the examination of the basal nerves reveals no abnormity.

In one case, although facial paralysis existed, the facial nerve, which was imbedded in the moderately infiltrated pia, showed no change in its tissue; the ventricular effusion was large, but there was no compression of the facial. The cause of the paralysis must, therefore, have been intracerebral. In another case the paralyzed facial showed a thick covering of purulent deposit; the nerve was thinner than normal, and tinged yellow. There was a great deal of cell development in the nerve sheath (migration?); the fibres were apparently intact. In the nervus acusticus different appearances were also found—sometimes inflammatory changes, and sometimes a normal condition. In other cases extravasated blood was found on and in the nerve. This we have not ourselves observed. Whether miliary tubercles have anything to do with it or not is not known. In one case the N. abducens was found greatly diseased in a case of strabismus convergens (there was injection and diffuse suppuration between the fibres). We found the same pathological changes twice in the oculo-motorius (strabismus divergens, dilated pupil, ptosis). In several cases of partial paralysis of the oculo-motorius there was nothing to be found in the nerve, and the disturbance of function could only be attributed to the brain. We have had no acquaintance with the changes of the trochlear. The trigeminus showed no abnormity in any carefully examined case. We have always found the opticus in a normal condition, though in a few cases there were evidences of moderate compression; the sheath, however, may sometimes be involved. A compression of the vena ophthalmica in such cases is highly probable; but this must be of subordinate importance in its influence upon the condition of the circulation of the retina. The sheath of the opticus in a majority of cases, where there is a good deal of internal pressure, is raised in ampullæ near the bulb, and a small quantity of serous fluid is found between the sheath and the opticus. In regard to neuro-retinitis, congestive papilla, etc., see further on.

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[1] Deutsch. Archiv f. klin. Medicin. V. Bd. 1867.

Some exceptional appearances are worthy of particular mention

1. Circumscribed œdema of one cerebral hemisphere conjoined with hemiplegia during life (Colberg).

2. Localized disease in the substance of one hemisphere, with paralysis of the arm during life (Traube).

3. Thrombosis of a sinus resulting from a thrombosis of the veins of the pia (Griesinger, reported by Seitz, p. 288).

4. Formation of cheesy tumors in the substance of the brain, from the transmission of a chronic tuberculous process of the pia upon the substance of the brain (Biermer, quoted in Seitz).

5. A large apoplexy in one hemisphere with rupture into the ventricle (personal observation).

6. Formation of chronic thickenings of the pia occasioned by circumscribed tubercular deposits.

All these are evidently the results of a tuberculosis of the pia, the course and extent of which may vary greatly. A few anatomical changes are also sometimes seen, which must have existed before the formation of miliary tubercles in the pia; as, for example, caseous deposits in the cerebellum (Biermer, quoted by Seitz, p. 161), terminal processes of tuberculosis of the pia, resulting either from this or another primary caseous spot. In one of our cases (narrated further on) there was a cheesy spot in the occipital lobe, and from this originated a limited miliary tuberculosis of the pia, without tubercles existing elsewhere in the body.

Spinal cord.—Our knowledge here is quite fragmentary. It is certain that tubercles are found in the spinal cord in many cases of tuberculosis of the pia, and also that their behavior is the same as in the pia of the brain. The inflammatory affection of the pia seems to pass down a varying distance within the canal. There are no trustworthy statements as to the changes of tissue in the spinal cord; but without doubt many symptoms would, after a more careful investigation of this subject, appear in an entirely different light from that in which they do now.

II. It is important to keep in mind the changes in the other organs:

1. *Organs of respiration.*—Larynx: miliary tubercles on the vocal cords; various degrees of old tubercular ulceration; chronic and acute catarrh of varying intensity, with follicular swelling. Trachea: Catarrh of all degrees; miliary tubercles on the mucous membrane. Pleura: old adhesions; old foci of cheesy deposit; fresh miliary tubercles; exudations, either fresh and serous, or with more or less addition of cellular elements, in some cases even reaching the condition of a thick purulent secretion (the latter varying much in character), often with tracts of old "pyogenic membrane." Lungs: in some rare cases were in quite normal state (7 : 130, Seitz); in another series of cases were affected with miliary tubercles throughout (41: 130); a very large number of cases showed the changes which are commonly spoken of as phthisical—alveolar infiltration in different stages of cheesy metamorphosis; peribronchitic changes in all stages of ulceration; bronchiectases of all kinds; cirrhotic thickenings and cicatrices—all either with or without recent miliary tubercles, though these are oftener present than not. Bronchial glands: a surprisingly great number of cases show cheesy degeneration of the bronchial glands; slaty induration and calcification frequently occur, but without relation to the miliary tuberculosis.

2. *Heart.*—The changes are partly of an accidental nature: old heart lesions, and obliteration of the pericardium; others have more or less relation to the miliary tuberculosis of the pia mater, such as old pericarditis, with cheesy degeneration; miliary tubercles in the pericardium, with or without inflammatory changes; hydropericardium; finally, fatty degeneration of the muscular substance of the heart.

3. *Intestine.*—This shows the least constant changes: miliary

tubercles in the mucous membrane of the stomach; old ulcers of the stomach; catarrh of the small and large intestines, with follicular swelling; tuberculous ulcerations, and diphtheritic ulcers.

4. *Liver.*—This shows occasionally miliary tubercles, and, in addition, all kinds of irrelevant changes.

5. *Spleen.*—The spleen is enlarged in more than half the cases. In one case it contained miliary tubercles; but these were not found in a large number of cases where the spleen was enlarged.

6. *Peritoneum.*—Here were found: miliary tubercles, without effusion; miliary tubercles, with inflammatory effusion; old peritonitis, with its products undergoing cheesy degeneration; finally, peritonitis from accidental causes.

7. *Pancreas.*—Foci of cheesy degeneration have been found in it, as well as miliary tuberculosis (Andral).

8. *Uro-genital organs.*—Miliary tubercles of the kidneys; cheesy metamorphosis and ulceration of the pelvis of the kidney and of the kidneys themselves; cheesy foci in the walls of the bladder and of the ureters, in the testicle and the epididymis; perinephritic abscesses, with cheesy degeneration.

9. *Lymphatic glands.*—Cheesy degeneration of the mesenteric or retroperitoneal glands, the cervical, axillary, and those in all other sites.

10. *Bones and periosteum.*—Suppuration of the vertebræ in every stage. We met with a whole series of cases of this in which no old cheesy affection of the lungs existed, but a general miliary tuberculosis; suppurations of the bones of the extremities and of the pelvis; affections of the periosteum, and especially of the joints (ribs, humerus; caries of the elbow, shoulder, knee, ankle, and hip joints); caries of the nasal bones from syphilis, ending in phthisis; caries of the clavicle, sternum, and petrous portion of the temporal bone.

Such an enumeration can only be of very limited value. The study of many individual cases can alone bring one to a knowledge of their connection. A conclusion then suddenly forces itself upon the observer, which has been a thousand times confirmed, and is daily asserting itself, *that in the immense majority* *of cases of tuberculosis of the pia mater a cheesy focus exists in the body*, whether in the lymph-glands, in the lungs, in the bones, in the genitals, or in the peritoneum. It is best from the outset to fix in mind the idea *of a primary affection of a cheesy nature*, and to speak of *a chronic inflammation which, by virtue of an existing constitutional anomaly, has the tendency to form cheesy products.* Moreover, it must be noticed that in very many cases the development of tubercle in the pia mater does not follow immediately, but *after an intermediate affection of a second organ, which is very commonly the lungs;* or that at first outbreaks of tubercle may occur in quite remote parts of the body, and only later in the pia mater, so that the two attacks are at quite distinct periods. Hence, in cases of caries of the vertebræ it is decidedly less common for the tubercles to occur immediately in the pia mater; but they develop first in the lungs, there excite the well-known secondary morbid changes, with cheesy degeneration and ulceration, and afterwards appear in the pia mater of the brain. A small number of cases fail to show such a primary focus. These will be spoken of later.

We are acquainted with the following causes, to which the development of miliary tuberculosis can be clearly traced.

Bones and joints.—Caries of the vertebræ and of other bones; consecutive miliary tuberculosis of the lungs, with the resulting changes: miliary tuberculosis of the pia. Affections of the joints, with the same results. Caries of the vertebræ, with consecutive general miliary tuberculosis, and also of the pia. In this case no intermediate eruption of tubercle. Caries of the hip-joint, with consecutive miliary tuberculosis of the peritoneum; chronic inflammation of the latter, with products which undergo cheesy degeneration: miliary tuberculosis of the pia. It sometimes, indeed, happens that the lungs also become diseased in such cases before the pia, so that there are two intermediate eruptions, and there may be even more, which run their course in organs of less importance, or which are usually less prone to suffer from tuberculosis than the others. Caries of the sternum, with consecutive miliary tuberculosis of the pleura, with purulent, gradually inspissating exudation: general miliary tuberculosis of the whole body. These examples by no means exhaust the combinations that occur.

Pleura.—Purulent pleuritic exudation, with thickening, the lungs being quite normal: consecutive miliary tuberculosis of the whole body, or of the pia alone. Purulent pleuritic exudation, with consecutive miliary tubercles in the lungs or peritoneum, or in both at once, with local secondary morbid changes: miliary tuberculosis of the pia.

Lungs.—Primary miliary tuberculosis, with consecutive phthisical changes of every kind; resulting pleuritis of various nature, or tuberculosis of the peritoneum with chronic inflammation; tuberculosis of the intestines, with formation of ulcers, with tuberculosis of the abdominal organs in every possible combination; finally, with consecutive disease of the bones and joints (caries of the petrous portion of the temporal bone; caries of the ribs, etc.): general fresh miliary tuberculosis of the whole body, or of the pia mater alone. Bronchial affection, with secretion undergoing cheesy changes; ulceration extending into the parts where there are cheesy foci: miliary tuberculosis of the neighboring tracts, which leads to further cheesy degeneration: general miliary tuberculosis, and so on.

Pericardium.—Chronic pericarditis with exudation becoming caseous; consecutive miliary tuberculosis of the pia, or intervention of a secondary affection of lungs or pleura; or widespread cheesy degeneration of glands.

Peritoneum.—Chronic peritonitis, with cheesy changes; consecutive tuberculosis of the intestine, with ulceration, or the same, in reversed order; consecutive miliary tuberculosis of the lungs, with chronic changes; finally, general miliary tuberculosis, or tuberculosis of the pia alone. Disease of the glands may come into the series of morbid changes in various ways. An affection of the pleura may also intervene, or the miliary tuberculosis of the pia may follow immediately on the cheesy peritonitis.

Perinephritic abscess, with cheesy degeneration; general miliary tuberculosis.

Uro-genital system.—Development of ulcerating cheesy foci, thence miliary tuberculosis of the lungs and pleura, or of the peritoneum; or, on the other hand, secondary disease of the latter after that of the lungs: general miliary tuberculosis.

Glands.—Primary cheesy degeneration of the bronchial glands; general miliary tuberculosis or affection of the pia alone; or between the two an intervening attack, or a whole series of them (miliary tuberculosis of the lungs with phthisis, pleuritis, peritonitis, etc.); or a cheesy degeneration of other glands, both deep-seated and peripheral, follows on that of the bronchial glands; and, later, general miliary tuberculosis. Primary cheesy degeneration of the mesenteric glands, and primary disease of the retroperitoneal glands.

Brain.—Tubercles of the brain, and then circumscribed tuberculosis of the pia mater, confined to the neighborhood. All these combinations and many more of less importance have been observed.

Over against the above cases stand a smaller number, in which the absence of a primary focus has been surely established. These cases remain for the time unexplained. One does not willingly entertain the theory which has been already promulgated, namely: that if miliary tuberculosis be a disease due to the resorption of a certain material, the matters absorbed (the centre from which absorption has taken place) may indeed disappear, and under certain circumstances must necessarily disappear.

9.—Boy of seven years; coxitis at the age of one and a half years, with favorable course and quick recovery. Till seventh year entirely healthy, without any affection of glands or joints. Middle of June, 1875, typhoid with grave affection of the lungs, lasting five weeks, afterwards a short relapse; great weakness after the typhoid, but for three weeks constant increase of strength. Intelligence normal, but less gay and lively; disposition to solitude, to entire seclusion; unfriendly, morose behavior. October 10, 1875.—Beginning of a new illness.

10th.—Headache, giddiness, disposition to sleep, quiet night, but, on the 11th, head symptoms more serious, chills, severe headache, heat, drowsiness, sensitiveness to light, kept the eyes constantly closed, and could not be persuaded to open them; quiet night. 12th, 13th, and 14th October.—Same condition; intelligence normal, but severe headache, giddiness, sopor, sensitiveness to light, appetite good, indeed immoderate, but on each of these three days severe vomiting—constipation. On the 15th and 16th no more vomiting, but great weakness and fatigue; constant disposition to sleep, headache, and constipation. On the 17th, worse in every respect;

patient can hardly be aroused, cannot sit up, eats nothing, is sometimes delirious. On the 18th the same condition, exaggerated; never quite in his right mind; almost the whole day in a state of sopor. On the 20th, carried to the hospital. Slight frame; emaciation; patient lies in deep slumber; quite irresponsive; from time to time slight groaning; no facial motor phenomena; no ptosis; eyeballs turned towards the left, the left eyeball more than the right one; pupils very sluggish; moderately dilated, equal; trismus; all the extremities in a state of violent but variable contraction; the spasm occasionally ceases entirely; unable to stand; contraction of muscles of the back of the neck; but this too of varying intensity. Breathing hurried, irregular; belly retracted; slight erection of penis; on slight irritation of the skin follow marked and extended red patches. Spleen not enlarged; heart sounds clear; lungs show nothing abnormal; temperature. 37.2° to 38.6° [98.9° to 101.5° F.]; pulse 80 to 104; no vomiting; no movement of the bowels; passes urine in his bed. 21st.—Ptosis on the right side; both eyeballs turned towards the right; slight divergence; no facial motor phenomena; contraction of muscles of the back of the neck. Trismus; entirely irresponsive; sopor; breathing irregular; meningitic spots. On passing a catheter through the nose, for the purpose of feeding the patient artificially, all the vessels of the face are suddenly dilated and tears come; spleen not enlarged; lungs show nothing abnormal. Temperature 37.2° to 38.2° [98.9° to 100.8° F.]; pulse 96 to 136. Examination of retina (Prof. Horner) shows: no tubercles on the choroid; papillæ in the first stage of slight swelling, with stasis in the veins and in the distended smallest vessels of the papilla; outline not quite clearly defined; slight cloudiness of those parts of the retina immediately surrounding the optic nerve. Right oculo-motor paralysis; hence the divergence. 22d.—Paralysis of left facial; otherwise everything in same condition; temperature 38.0° to 37.4° [100.4° to 99.3° F.]; pulse 120 to 92.

23d.—Nystagmus of left eye; left facial paralysis more marked; tongue wedged between the teeth, and can only be replaced with difficulty; many quickly recurring short attacks of opisthotonus; urine without albumen (1019, sp. gr.); temperature, 36.0° to 37.8° [96.8° to 100.4° F.]; pulse 92 to 100; irregular.

24th.—The same condition; but now all the extremities relaxed, without resistance; neither contraction of muscles of the back of the neck, nor retracted belly, nor convulsions; eyeballs turned towards the left; diverge more than before; no nystagmus; pupils moderately wide; not responsive; breathing irregular. Death occurred in the evening, with a marked rise of temperature in the agony—37.6° to 38.2°, 41.4° to 41.6° [99.7° to 100.8°, 106.5° to 106.9° F.]; pulse 120, 128, 136, 142.

Autopsy.—Some scattered miliary tubercles in the dura mater; a large amount of exudation at the base; a very trifling infiltration of the pia visible to the naked eye; in this last a general scattering of miliary and submiliary tubercles. Marked hydrocephalus internus with compression of the brain. Left pleura shows miliary tubercles; both lungs the same. In the liver and kidneys some few tubercles; spleen enlarged, without tuberculosis; all the glands entirely normal; nothing to be found either in the bones, periosteum, or marrow; no primary focus. (My own observation.)

In the above case the disease appeared three months after typhoid ; in another case two months ; in a third case one and a half months. The development of miliary tubercles in the pia mater must have taken place before the beginning of the acute attack, which last coincides with the commencement of inflammation of the pia.

Similar cases have been reported by Biermer (See Seitz, Case XV., where nothing was found in the cadaver beyond an old adhesion of pleura and pericardium, and a calcification of bronchial glands of the size of a grain of hemp-seed ; and Case XIV. —a similar calcification, together with a small ovarian cyst, were all that was found), and also by Wilks :

10.—Girl eighteen years old; always healthy; eighteen days before death complained of her head ; on eighth day of the disease admitted into the hospital as a case of typhoid ; in a state of sopor ; lies on her side, drawn up in cramped posture ; hyperæsthetic when moved or touched ; ptosis ; both pupils dilated ; sluggish ; during the last two days preceding death, coma ; difficult breathing ; no convulsions. *Autopsy.*—Purulent exudation at the base and on part of the convexity ; numerous tubercles in the pia mater of both hemispheres ; ventricles showing abundant effusion ; no sign whatever of any primary focus ; no miliary tubercles elsewhere.

These rather unusual observations have not yet found their explanation ; we cannot but think that the primary focus, really existing but in an unusual place, may have been overlooked Decidedly, however, these observations cannot throw doubt on the truth of the maxim that in the very great majority of cases of tuberculosis of the pia mater (and of the whole body) a primary focus of yellow cheesy degeneration is to be found. In consequence of this, the greater number of physicians are accustomed to consider miliary tuberculosis of the pia mater as part of a general specific disease of absorption or infection, which has the same relation to the cheesy foci "as pyæmia has to a centre of decomposition, or acute miliary carcinomatous affection to an original cancerous formation." A poison, entirely *sui generis*, tubercular matter, comes from this focus into the blood, and "is deposited (as variola poison is in the skin) in innumerable places, in the form of gatherings, varying in size from a kernel of wheat to a poppy-seed, composed of easily degenerating nuclei and cells"

(Buhl). Further, it is thought that, to cause the disease, the very minutest quantity of this material would be sufficient. Its form would not be the characteristic of this tubercular poison, but its chemical composition, which has thus far not been discovered. The absence of a primary focus in some cases might be thus explained.

Thus far we have not advanced much beyond this point of view; what follows may perhaps serve to bring us nearer to the reality.

Doubtless there is something, which somehow finds its way from the cheesy foci into the circulation, and, reaching various places in the tissue, excites there the development of the tubercle. If we seek to discover the way in which this substance is distributed in the body, we encounter at once the hitherto unexplained difficulty, that the method of absorption is only so partially known as to make us hesitate to give any explanation of the remarkable spreading of the miliary tubercles. Let us ask, further: What is absorbed? Is it simply tissue juice from the focus? Is it small particles of the tissue which find their way into the blood? Is it perhaps both of these, forming a sort of emulsion, in which the particles of disorganized tissue are held in suspension? Does this infectious matter, then, find its way into the blood, into the lymph, or whither?

The ways in which it is distributed are certainly various; it is certain (Virchow, Rindfleisch, Klebs) that a distribution is possible through the lymphatic system; and this certainly often takes place in many regions, and especially in the neighborhood of cheesy foci. The lungs present very favorable conditions for this; likewise the intestines and peritoneum, if neighboring lymphatic glands are diseased.

But in the pia mater the spread and mode of origin of miliary tubercles do not seem to authorize the conclusion that the lymphatics have brought the infectious matter. Let us observe, in the first place, that tuberculosis of the pia mater is by no means always complete—indeed, that in individual cases the spread of the miliary new-formations does not go beyond the tract of a particular artery. It happens, for instance, that miliary tubercles exist only in the tract of the anterior branches of the circle of

Willis, or only in the tract of three branches, or, finally, in that of only a single branch; in this latter case, according to our information up to the present day, it is the artery of the fissure of Sylvius, and more particularly on the left side. In such cases a formation of pus, visible to the naked eye, occurs only in the tract of this one artery; and the pathological changes are then limited, in the majority of cases, to the formation of tubercles and of pus, and hydrocephalus is absent.

Further, we have seen cases in which tuberculosis was only developed in the tract of the posterior arteries of the brain, arteriæ profundæ, and the cerebellar arteries; the infiltration of the pia mater in these cases was much more extensive, and hydrocephalus was not wanting.

11. Man, nineteen years old; small-pox two years ago; typhoid one year ago; since that time weakly and reduced, without well marked symptoms of local disorder; nothing visible in the external lymphatic glands; he will not acknowledge to any lung symptoms. Duration of disease seven days, beginning with general undefined illness, shortly followed by severe headache, giddiness, and delirious attacks. Excited condition, abusive language, cries, attempts at escape, so that patient is considered to be insane; pupils contracted; fever; pulse rapid.

On the morning of the second day quieter; from time to time head entirely clear, with intervening short periods of sopor; headache; giddiness; fever. Gait and speech not affected, neither paralyses, nor other of the more important motor symptoms. Towards close of second day recommencement of the delirious attacks, excitement, hallucinations in the sense of hearing, attempts at escape, etc. It was noticed at the same time that the patient moves the left arm less than the right; no difference noticeable in the lower extremities. The third day passes with constant delirious excitement, cries, noise, raging, resistance to all rules and regulations. Left arm still more paralytic; left facial paralysis; left pupil wider open and less responsive; hypoglossus not affected; no strabismus; no ptosis. Fever; pulse 120, small but regular. Mucous râles in the left apex; in the right upper back bronchial expiratory sounds. On the fourth day, in addition to the existing paralyses, there appears *strabismus divergens* (paralysis of left rectus internus); the left pupil very widely open (no ophthalmoscopic examination); paralysis of left side of face, and of left arm; no paralysis of the leg. Excitement less, but no sopor; patient distracted by troubled dreams; does not recognize those about him; does not know where he is. Vomiting; no contraction of muscles of the nape of the neck; abdomen not retracted; constipation. On the fifth day frequent alternations of excitement and quiet; fever runs higher; pulse 116, somewhat irregular. Paralysis the same; weakness more general; emaciation. One attack of vomiting; constipation; no contraction either of the muscles of the back, or of the muscles of the abdomen; no convulsive symptoms; speech continues distinct. On the sixth day moderate fever; pulse 128–136, small and irregular; total left oculo-motor paralysis; *ptosis on the right side* appears all at once; there is left facial paralysis and paralysis of the left arm. Drowsy condition; no excitement; no sopor. Death occurred on the morning of the seventh day with marked rise of temperature, the pulse dwindling and increasing in frequency till it could no longer be counted.

Autopsy.—Nothing of importance in the skull or dura mater. Base of the brain shows a tolerably widespread yellow exudation, *greater on the right side than on the left.* The right oculo-motor enveloped in a sheath of purulent exudation (ptosis on the sixth day); left oculo-motor nerve normal to the naked eye (its total paralysis was, therefore, caused by some lesion within the brain). The exudation and suppuration follows the right fissure of Sylvius and extends quite to the convexity. The courses of all other arteries are entirely free from suppuration. There is no actual compression of the brain, only very trifling hydrocephalic effusion; no softening. The artery of the right fissure of Sylvius has implanted upon its finer ramifications a very great number of miliary tubercles; some of these are found as far as the longitudinal sulcus, backward as far as the occipital fossa, forward even beyond the anterior central convolution. No tubercles to be found on any other artery; the left fissure of Sylvius is entirely free from them. (My own observation.)

Below will be given some further cases of isolated affection of one arterial tract. What these cases teach us is of importance, since it fortunately happened that the examination showed:

a. Tuberculosis in the tract of a single artery of the brain.

b. Far from any general miliary tuberculosis, tubercles were *only* found in the pia mater.

c. A primary cheesy focus with softening in places, so that an explanation offers itself as to the manner of mingling of the infectious matter with the normal fluids of the body, and as to its mode of transportation. In this connection the following observation is instructive:

12. J. K., twenty-four years old, died of tuberculosis of the pia mater and of the whole body, which neither in its course nor at the autopsy showed anything unusual. Widespread phthisis. But the examination of the lungs points to one instructive fact. We find that the wall of a tolerably large pulmonary vein is perforated; the perforation is of the size of an apple-seed, and leads into a cavity, formed according to the usual process from a bronchus; a cavity as large as a bean, with ulcerated walls, and containing inspissated, purulent, cheesy masses. Many other such cavities exist in the lungs, but no other is found to communicate with a pulmonary vein.

The supposition, therefore, seems allowable, that a direct mechanical mingling of emulsive cheesy substance with the blood of the pulmonary veins is one of the ways in which the infectious matter arrives in the circulation. In regard to the tubercles distributed generally throughout the body—in regard to the discrete formation of the same in one of the smaller arterial tracts (the supposition seems especially allowable in regard to one of the arteries of the pia mater)—it would seem that we have to do with an embolic distribution of injurious matter which has found its way into the blood of the left heart. The theory of the occurrence of embolic processes has been, it is true, utterly rejected by many: we should, however, really like to ask how otherwise to explain those cases in which tubercles of the same age exist on the peripheral ramifications of one artery of the pia mater and nowhere else, in which the rest of the body is free, and in which finally there exists a cheesy focus showing in its interior some liquefied parts. The above observation very clearly shows the possibility of a mingling in the lung of the infectious matter with the blood of the left heart. This is, after all, only an attempt to explain those cases in which pulmonary phthisis forms the point of departure of the infection.

Further, the manner of transportation of emboli might be exceedingly various. For in one case all the miliary tubercles have evidently been implanted at the same moment, in another case successively, which last could be explained by an occurrence in the lungs like that mentioned above; the wall of a vein of the lungs, once perforated by ulceration from within, allows blood to escape into the cavity; at first very little becomes charged with injurious matter; little by little the perforation grows larger, until at last the cavity is literally washed out by the stream of blood. In one case there are only very few miliary tubercles, in another an innumerable quantity; and this again may be explained by the manner of the mingling. But, finally, the distribution of miliary tubercles may very readily happen in still another manner. If, in the beginning, there exist only a few, so that no general inflammation of their matrix follows, they may then run through their metamorphosis into fatty granules, and new tubercles may take their rise from them by way of the lymphatics of the pia mater; this manner of distribution occurs perhaps with great frequency.

Explaining the matter by the theory of the entrance of cheesy masses from the lungs into the blood of the left heart, does not, however, by any means exhaust the subject. How, for instance, does infectious matter find its way into the arterial circulation when nothing is found in the lung beyond a number of miliary tubercles, which have the same age and degree of development as the miliary tubercles of the pia mater?

13. Girl, fifteen years old; illness of nine days' duration, following in every respect the usual type.

Old cheesy degeneration of the bronchial glands, some of them showing puriform softening at the centre. Lungs showing not the slightest trace of any old process. Both lungs contain, however, a relatively small amount of miliary granulations, all of the same appearance and age; heart, pleura, peritoneum, abdominal organs without any miliary tubercles; tuberculosis of the pia mater with the usual anatomical appearances; marked hydrocephalus. (My own observation.)

And, in opposition to these common results of observation, are those more unusual ones where a primary cheesy focus is found, while the lungs are entirely free from miliary tubercles.

One is therefore forced to the supposition that—in addition to the mingling of the contents of softened cheesy foci in the lungs with the blood of the left heart, in addition to the local distribution through the lymphatic ducts of an organ—still other ways must exist, by which the infectious principle can be distributed through the body. It does not advance us to discuss here the tenability of the supposition that it is possible for the venous system to take up some element from softened, cheesy lymphatic glands, and to carry this substance to the lungs. It is impossible to decide the question, whether it happens that a portion of what is carried to the lungs is held fast there, while another portion can pass through the lungs and reach the arterial circulation, although there are cases enough of miliary tuberculosis of the lungs, which, on account of the equal distribution of the miliary tubercles, and of their exactly equal age, can scarcely be otherwise explained than by the transportation of the cause by the blood. Finally, it is impossible here to go into the question what part is taken by the great lymphatic ducts in the impregnation of the blood with elements which have their origin in cheesy glands. It is probable that all these ways of infection exist. But we repeat, once again, that there are cases of miliary tuberculosis of the pia mater, *for the explanation of which one can only invoke a multiple embolism of the arteries of the brain.*

It has already been hinted at above that it is still a question in regard to the *successive* formation of tubercles in the pia mater, whether the propagation of the new-formation, proceeding from a limited number of tubercles which are in process of fatty degeneration, may not follow the course of the lymphatics. In the following case an old cheesy focus existed on the surface of the left occipital convolution, and in its neighborhood a relatively small number of tubercles, which, without doubt, owed their origin to that primary focus.

14. Factory girl, sixteen years old; during the last two years phthisical infiltration and formation of cavities in both apices; is said to suffer from attacks which have been called epilepsy; no other nervous symptoms than frequent headache; no further sensory and no motor derangements. Glands of the neck suppurating; death after an attack of copious hæmoptysis, without remarkable nervous appearances.

Cheesy focus the size of a hazelnut on the surface of the convexity of the left occipital lobe, at the point where the occipital fissure intersects its surface. Around this focus is an edging of yellow necrotic softening. The pia mater in the neighborhood is considerably thickened; there is, however, no suppuration, no exudation at the base, no hydrocephalus. About the cheesy focus—traceable posteriorly as far as to the point of the occipital lobe, anteriorly about as far as to the anterior central convolution—were a great quantity of miliary tubercles. No noticeable affection of one or many arterial tracts could be proved; all the basal arteries and the right hemisphere were entirely free. (My own observation.)

Here hardly any other supposition is possible than that of a local distribution through the lymphatic ways. This case may, at the same time, serve to call attention to the fact *that miliary tubercles may be developed in the pia mater without a single symptom during life leading us to suspect their existence.* The symptoms of the acute disease are occasioned by the inflammation of the pia mater and of the cortex, and by the product of this inflammation, and are not occasioned by the developmen of the miliary tubercles.

After these explanations of the cause of the disease, which are unfortunately, from the very nature of the case, so incomplete, let us turn to a consideration of that more remote etiology, which, in regard to the disease we treat of, is of fundamental importance, as it is in regard to all diseases arising from tuberculosis. The belief is not ungrounded that certain individuals show a tendency to the formation of cheesy foci, which so often cuts them off in early youth, while this tendency seems to be entirely wanting in the case of other individuals.

Tuberculosis is built up almost without exception upon scrofula as a basis. Pathological anatomy shows it to be characteristic of scrofulously diseased assimilation that almost all inflammatory processes show no inclination to absorption nor to an active formation of pus, but, on the contrary, a tendency to retrogressive metamorphosis, which, to the naked eye, offers the appearance of cheesy degeneration. The reason of this behavior is not yet explained ; let it suffice to mention the long-proved fact that the product of scrofulous inflammation is exceptionally short-lived, a fact which seems to hinder a normal elimination of the cellular elements. Rindfleisch gives it as his opinion, considering the size of the cells of the product of scrofulous inflammation, that these have a tendency to increase in size on their way from the vessels through the cellular tissue, to swell by intussusception of substances rich in albumen, but meanwhile, during this process of swelling, to die, and gradually to disintegrate where they are.

Let this be as it may, it is at all events certain that the scrofulous infiltration has a character entirely its own ; that in it takes place neither the normal purulent melting, nor an absorption, nor an organization into cellular tissue ; on the contrary, it stiffens the cellular tissue into a tough, compact mass, gray by transparency, rendering impossible the passage of blood through the blood-vessels. Thus is the alimentation of the infiltrated substance suspended, and this undergoes a sort of dry necrosis, which appears to the naked eye as a cheesy degeneration, beginning in the centre and advancing outwards. Then it is not necessary that there should be great cheesy lumps in certain localities, which cause the tuberculous diathesis ; but almost any part of the body is capable of furnishing a material which may afterwards come to be absorbed as a self-formed poison. The mucous membranes are of importance. We can no longer doubt the existence of a characteristic scrofulous affection of the mucous membrane, which fills, for example, the adenoid tissue of the intestinal mucous membrane with disintegrated exudation cells, which are then reabsorbed by the lymphatics. The result is tuberculosis of the mesenteric lymphatics and tuberculosis of the mesenteric glands. Of the greatest importance in this connection is the proof (Schüppel and Rindfleisch) that the infiltrated lymphatic glands of scrofula contain already the fully developed miliary tubercle. When, therefore, Rindfleisch speaks of a secondary tuberculosis, meaning by this the affection of the lymphatic glands, no sound reason can be brought forward in opposition to this. Tertiary tuberculosis would then be the appearance of miliary tubercles disseminated throughout the body; consequently, in the pia mater as well. But, finally, what is that which is absorbed? A softened cheesy focus contains a number of nuclei, solitary and agglomerated, which consist of an unknown modification of albumen; then there are masses of small fatty drops; then small scale-like elements, with irregular border, and without visible organization; finally, a liquid the quality of which is entirely unknown to us. These substances, perhaps all of them, reach the vascular system. We may suppose that in one case they find their way thither slowly and successively; that they often are held fast in the lymphatic way which they have reached, developing there (formation of lymphatic knots in lymphatic vessels and glands) their disastrous activity, and then giving a further secondary opportunity for cheesy degeneration and absorption. Thus a tuberculosis may slowly and successively invade the body. We may, however, also suppose that from a cheesy focus the substance in question reaches the venous circulation in greater quantity, and then the infection of the lungs is the direct result; by this we must not be understood to say that this is the only way in which the lungs are infected.

Finally, in accordance with what has been already explained, we are unable to deny the possibility that, by way of the arterial circulation, after previous mingling in the tract of the pulmonary veins, these substances, besides reaching other organs, may reach the brain too, and may there excite a growth of miliary tubercles. The formation of cheesy foci causes an accompanying cachexia, which we find in the majority of those individuals who are attacked by tuberculosis of the pia mater.

Now it is still entirely unknown which ingredient of the softened cheesy mass works as the infectious agent; the manner of impregnation of the endothelial structure is involved in an equally deep obscurity. Do poisonous elements find their way from the vessels into the pia mater? Are these really things to be seen with the eye, or is it a liquid, or are they elements of the blood which have acquired a certain character in the cheesy foci? These are all questions which await an answer.

Scrofula is therefore to be counted as one of the chief etiological factors in tuberculosis of the pia mater. Experience in thousands of cases proves that in the far greater majority it is scrofulous individuals, or those who have been scrofulous, who are attacked. It is not necessary that the individual should have the outward marks of scrofula; this, however, is necessary: that in him should exist that tendency to bring inflammatory exudations to a cheesy degeneration. It is then easily understood that all sorts of acute diseases which cause a great number of inflammatory lesions lead, in the case of individuals with a disposition to scrofula, to dead cheesy exudations, instead of leading to such as simply suppurate, or become indurated, or are absorbed. Whooping-cough and pneumonia, or catarrh following measles, simple catarrh, and broncho-pneumonia of children, are then capable of rendering the bronchial glands *cheesy*—it would be better to say *tuberculous*—by the absorption of degenerated exudations. Catarrh of the small intestine receives, in the case of a child with this tendency, a specific modification, so that from this begins tuberculosis of the mesenteric glands. Typhoid can deposit in the bronchial mucous membrane and in the lungs infiltrated matter, which leads to the same result in regard to the bronchial glands. But traumatic lesions of the extremities, of the bones, of the periosteum, of the joints, lead to the same result, since the inflammatory exudation, in the case of a person with this tendency, enters upon the abnormal transformation already mentioned. It is not sustaining a paradox to say that miliary tuberculosis of the pia mater may be caused by a blow or by a spontaneous inflammation of the periosteum of the extremities.

Psychical influences are repeatedly mentioned as etiological forces.

Two cases have come to our observation, in which, after excessively intense depression of the spirits, there followed an indefinite general illness, soon leading to febrile brain symptoms, from which the whole sequence of symptoms of tubercular meningitis was developed. In one case, in which the interval between the psychical affection and the beginning of the disease lasted about fourteen days, the correctness of diagnosis was proved by the autopsy. Until the occurrence of the moving cause, there existed relatively good health. The primary focus was old latent tuberculosis of the lungs, with some fresh cheesy degenerations, and with them miliary tubercles of the lungs.

In regard to the manner of action of the moving cause, we do not dare to give any opinion. Similar cases are reported by Hessert and Oppolzer.

In some cases traumatic accidents preceded, and stood in such evident connection with, the beginning of the illness, that it was impossible to deny to these the causative rôle (Finger, Wilks, Griesinger, given in Seitz, p. 23—the latter case, however, not very conclusive).

Intoxication seems to have been enough to give the impulse to miliary tuberculosis. Strong action of the heat of the sun on the head has been charged with causing the same.

Immoderate muscular exertion was evidently the moving cause in one case which we observed. After this followed hæmoptysis, and only a few days afterward the alarming nervous symptoms.

The irritation of dentition, diarrhœas, colds, play their part among children. It appears that frequently the moving causes of leptomeningitis infantum and of tubercular meningitis have been intermingled.

It may be concluded, from this enumeration of the etiological forces, that scrofula furnishes the chief contingent of cases of tubercular meningitis at every age. How mankind came to be afflicted with scrofula is not here to be considered; we merely mention that this disease is clearly, in the majority of cases, inborn and inherited, and that it then begins to develop its symptoms under the influence of unpropitious external conditions. It is generally allowed that in this respect the nutrition of children is of deep importance; deprivation of mother's milk, bringing up with unfit nourishment rich in starch; deprivation of good air for breathing, occasioned by bad condition of lodgings—all cause a slumbering tendency to be aroused, a tendency already existing, to make rapid progress; as soon as cheesy degenerations of the glands once exist, the man is in reality already tuberculous, and is exposed at any moment to tuberculosis of the pia mater.

The frequency of tuberculosis of the pia mater is very considerable; crowded populations and large cities present the greatest number of cases. The statement of Bennet is well known, that in London there are a third more cases of "hydrocephalus" than in other English cities. It is difficult to give accurate numbers; Seitz estimates that in the Canton of Zürich, for ten thousand living adults, three die of this disease; all cases under fifteen years would have to be added to this number. It has been believed that this disease was influenced by certain seasons, that it was more prevalent in the cold months, especially in those of early spring. Such a law, however, cannot be proved. It is generally stated that the disease is more frequent in men than in women. If we only look at the cases over fifteen years (Seitz), we find a slight preponderance of males (51.5 men, 48.5 women); as soon as we add to these the cases below fifteen years, the preponderance of males is much more remarkable. The time of life from two years to the beginning of the seventh year shows the greatest disposition (of two hundred and sixty-five of Bennet's cases, one hundred and eighty-three are below seven years); fewer cases offer from the sixth to the tenth year; the frequency sinks rapidly from ten to fifteen. A good point of reference, in regard to the time of life between sixteen and sixty, has been given in the statement above; from the forty-fifth year on the number of cases is only exceedingly trifling.

The disease makes its appearance most frequently among the lower classes of the people; still scrofula makes itself felt in all classes.

Finally, from all that has been mentioned, the influence of hereditariness, too, is plain. If we follow with attention the traces of scrofula, we shall frequently discover the influence of hereditariness. It is not tuberculosis of the pia mater which is inherited, but that tendency which causes its occurrence. It has often been observed that all the children of a family have died of tuberculosis of the pia mater. Still oftener one or two children die, and the other brothers and sisters drag themselves wretchedly through the world, with the various outward marks of scrofula, which in their own children first makes its appearance as true tuberculosis. Examples are known to every practitioner.

Symptomatology.

It has been a very old habit of authors to divide the progress of tubercular meningitis into several—generally three—stages, strictly separated one from the other. There was a certain excuse for this schedule arrangement, since there is a series of cases, which, in fact, in all their appearances, duration, etc., are very closely related, and allow a distinct separation of the various stages. One must, however, keep clearly in mind that there are just as many departures from this rule; these are caused by the very great variety of the patho-anatomical grounds, by the circumstance that of the various parts which make up the anatomical condition, one leaves the others far behind: hydrocephalus, for instance, quickly increases to such an extent that the brain is compressed before the basal exudation has reached such a degree of violence as would occasion paralyses in the tracts of the facial nerves; or, vice versa, the compression of the brain is very long wanting, while symptoms of lesions of the surface of the brain and also of the base come into the foreground.

The division of the progress of the disease into three stages has been formulated, with various divergencies, about as follows:

I. *Stage of brain irritation.*—Headache; vomiting; constipation; retraction of the belly; beginning of pulsus cephalicus; excitement and delirious attacks: in the case of children, convulsions.

II. *Stage of pressure.*—Pupil symptoms, states of sopor and coma; pulsus cephalicus; constipation; belly retracted; contraction of muscles of the back of the neck; facial paralyses, which are generally referred to the base of the brain; lesions of the facial, hypoglossus, oculo-motor and abducens; hemiplegia; contractures; convulsions; frequent changes in the general condition.

III. *Stage of paralysis.*—Coma; quiet, as a general rule; decrease of all cramp symptoms; increase of the paralyses; disappearance of contraction of the nape of the neck and of retraction of the belly; rapid increase of beats of the pulse, which grows irregular; variations of temperature up or down, denoting approach of the agony: death.

The duration of these stages has been noted by certain authors as extremely various: Traube specifies eight days as the usual duration of the first stage; of the second, less than eight days; the third lasts only a short time, about twenty-four hours or less.

We may in general terms refer the first stage to the affection of the pia mater of the convexity and of the surface; the second stage to the development of hydrocephalic effusion; the third stage to the gradual paralysis of the centres of the medulla oblongata. The symptoms of affection of the base are distributed throughout the first two stages, or first make their appearance during the second stage, and are continued into the third. It is impossible to place the convulsions according to any regular order; these appear, in fact, especially in the case of children, in all the three periods, and up to the present day it has not been possible to designate with exactitude their pathophysiological cause; it is probable that many circumstances concur to produce them.

We shall now give, first, a description of the whole course of the disease, according to some simple typical cases, and shall then add a description of some abnormities in its course.

We have above already given it as our opinion that it must be allowed that, in the great majority of cases, the first symptoms of an alarming nature are not furnished by the miliary tuberculosis of the pia mater, but by the inflammation of the meninges, which is occasioned by the tuberculosis. We must, therefore, not be surprised if we observe to be wanting that prodromal period which has been, especially in the case of children, described with such care. If the individual is relatively healthy before the breaking out of the meningitis—that is, if he is in no way troubled by existing cheesy foci; if these are latent and have been, up to this time, shut off from the circulation of the lymph and of the blood, which is exactly in accordance with their nature—then we observe that a prodromal period is often wanting, in carefully observed cases, and the disease begins with head symptoms of an inflammatory nature in the midst of apparently good health. The preceding development of tubercles in the pia mater has then caused no symptoms beyond perhaps a slight, hardly noticeable, derangement of the nutrition, or an increase in such a derangement already existing. In the case of sensitive individuals, such a derangement of the nutrition already leads, to be sure, to all sorts of discomfort, weariness, loathing, general entirely undefined illness, loss of appetite. Some observations on this point showed us an entire absence of fever until the beginning of the head symptoms; while others showed irregular, not high, states of fever, which could not be surely explained. It is difficult to say on what these differences depend; but individuality plays a great part, as in the case of all causes which excite fever. In one case the prodromal period was characterized by a melancholy turn, sadness, objectless anxiety—the reasoning power remaining meanwhile quite intact. In the case of children this initial period is often much more marked; they change their psychical behavior; they become languid, depressed, sad, abandon their usual employments; sleep often, though never long at a time; dream a great deal; in spite of their depressed condition, there are frequent complaints of headache and giddiness. Still these attacks are mostly of short duration. Accompanying these symptoms, there is a diminution of appetite ; the tongue is coated ; irregularities at stool ; constipation and diarrhœa by turns. Little by little slight febrile condition at evening, and indeed much oftener than in the case of adults, and quite as little permitting a sure explanation. The change in the entire disposition and in the outward behavior resulting from this is so remarkable that it must at once awaken suspicion, especially where moving causes exist. We must not, however, expect these disturbances in all cases ; often there is nothing whatever to be observed beyond a slight derangement of the nutrition, to which some fever may afterwards be added, followed, after a shorter or longer interval, by nervous symptoms. A suspicion, arising under such circumstances, is then increased in some cases by the observation of convulsive attacks, which may already now occur. After the attack, either everything returns to its former condition, or the convulsion now marks the beginning of the diffuse inflammatory disturbance of the pia mater. In regard to the duration of this period, our own experience, and a mass of statements of others, show such discrepancies that a decision can hardly be risked (one week, some weeks, two months, etc.).

The position of things is different if one of those clearly recognizable affections has gone before, which, according to experience, stand in near relation to tuberculosis of the pia mater. If this has arisen on pulmonary phthisis as a foundation, then all the marks of this latter would be present, and the affection of the brain is then, in many cases, only a partial appearance of a dissemination of miliary tubercles studding nearly the whole body ; the brain symptoms, too, owing to the rapidity of the course of the disease, frequently come only to a very partial development. In such cases nothing can usually be seen of prodromal symptoms ; the patient has, without tuberculosis of the pia mater, high fever ; he is, without this, emaciated and miserable ; he is, without this, sleepless and restless, and from time to time delirious, so that the beginning of the brain disease is often not noticed, and we are suddenly surprised by a facial paralysis, by an attack of entire unconsciousness, etc.

Similar circumstances prevail in affections of the pleura, of the pericardium, of the peritoneum, with cheesy products, in caries of the vertebræ, in tuberculosis of the urinary and genital organs, in every more extended inflammatory lesion, which, founded on scrofula, reaches its own special form of development. In a case of miliary tuberculosis of the pia mater, coming from cheesy products in the pleura, the patient had constant high fever with the character of hectic fever; miliary tubercles had long settled in his lungs; but no suspicion could arise of tubercles in the pia mater, until the patient had a violent headache with vomiting, and quickly sank into unconsciousness.

We may, therefore, really learn something from foregoing lesions, especially in the case of children; so far as this, that experience must make us attentive to the importance of the same. It is significant, if whooping-cough, long-continued bronchitis (especially on a scrofulous basis), measles, scarlatina, typhoid fever, uncomplicated broncho-pneumonia, pleurisy, pericarditis, have gone before, and if there follows a protracted incomplete convalescence. It is of the greatest significance if at such times there appear swellings of the lymphatic glands; if a chronic catarrh of the bowels will not yield to treatment; if a chronic bronchitis defies all measures; if an undefined cachexia, with emaciation and wasting of muscle, attacks children; and if, finally, the above mentioned changes also occur in the character and psychical behavior of children.

In accordance with what has been said, the real beginning of the meningeal affection must also appear under very various forms. In one case which came under our observation there was a chill denoting a rapid rise in temperature. In plain cases the beginning was marked by shiverings; great languor and discomfort, coming suddenly; sense of tightness in the head, quickly increasing to violent headache; giddiness; fear of light; loss of appetite; thirst; disturbed sleep; restless dreams. Attacks of giddiness usher in nausea and loathing, and then vomiting, after which some patients feel a slight lessening of headache and giddiness; vomiting is, however, by no means of constant occurrence at this early stage. At the same time there is constipation; but this may be wanting during the first days, and the stool may be entirely normal. Finally, febrile movements appear. These last are of very varying intensity, and it is entirely impossible to construct a curve of any constancy. In some cases there is bleeding at the nose, giving temporary relief. We notice at once that these are symptoms such as may occur in the case of any violent flow of blood to the brain, and doubtless they do indeed correspond to such an occurrence. Moreover, in the above-mentioned sequence of symptoms one or the other falls into the background, and the others predominate; thus sometimes the headache reaches an extreme violence, or there appear still other symptoms which are less usual at the very beginning: slight disturbance of the intellect, troubled, unsettled condition, want of attention, slow thinking, unfit answers, great apathy and vacancy, stupid staring; in the case of children an *attack of convulsions* very often opens the scene, or at least appears very quickly after the first symptoms. Isolated observers (Hasse) call attention to the fact that very serious symptoms of the original disease fall suddenly into the background on the appearance of the meningeal inflammation, or at least grow really less intense. In the case of phthisis, the cough, dyspnœa, and expectoration soon leave the front rank; the sweats cease; the number of pulse beats decreases; in the case of intestinal tuberculosis the diarrhœa stops. This is entirely correct.

Abnormal forms of the beginning will be spoken of below. With the above-described symptoms, whoever feels a division into periods to be necessary, may declare the first stage opened.

The chief symptoms of the following days—till the 8th, with variations on both sides—are:

Headache.—This stands in the foreground in the great majority of cases, and continues, with slight variations, constantly; of this the patient complains greatly so long as he has his senses; and much later, when consciousness seems long extinguished, his complaints take the form of signs. The patient often puts his hand to his head, rubs it, supports it, wrinkles his forehead, distorts his face, as in pain. Many lament and cry out from time to time. The whole head is in most cases the seat of pain, seldom only the frontal region. At the same time, it is not seldom that a noticeable hyperæsthesia of the skin has been observed on the head and the whole body; in one case this was confined to the body alone. Pressure from without does by no means increase the headache in the case of adults, but does increase it in the case of young children, for reasons which are not far to seek. With these latter the fontanelle is often very sensitive; its pulsations are strong; no bulging is as yet noticeable.

Vertigo, great sensitiveness to light and noise are always to be observed; patients have the feeling that everything reels with them, that they are falling in bed, or that they are being raised, or their whole surroundings seem to be moving about; on account of this the gait is often reeling and unsteady.

Psychical disturbances.—These are not always evident; often the patient is not delirious; his judgment is not disordered; he is not subjected to illogical impulses of the will; and yet he is unable to take part in conversation; he can hardly remember, cannot form consecutive trains of ideas; hence his slow answers, or often none at all, his want of quick perception, his unsettled and dreamy condition. This state often makes at this time very rapid progress, so that between evening and morning a very great advance may be observed in the ruin of the mental faculties. Delirium during wakefulness is not very frequent, but will be mentioned below as exceptionally occurring on a very extensive scale. Delirium is, however, of frequent occurrence when the patient is half asleep; in the case of children the hydrocephalic cry is at this time not unfrequently heard, accompanied by frightened startings up. Then come often hours of entire absence of the mind, in which the actual world has entirely vanished, so far as the patient is concerned, and he is sunk in a deep dreamy condition. Then, while the eyes stare without expression into vacancy, he talks senselessly with himself, or laughs, laments, counts, or the same words are unwearyingly repeated without expression, or the patient sings, whistles, shouts; all this accompanied by movements more or less senseless or meaningless. Many of these movements have clearly the character of entire want of participation of the mind; thus, for instance, the carphologia, the picking at the coverlid, repeated for hours together, the plucking at objects, the pulling at parts of the bed, the blowing, spitting, grimacing, etc. Other abnormal muscular movements show the character of a participation of the diseased sensorium; thus the defensive movements against hallucinations of the sight, the sudden springing from bed in consequence of deceptions of the ear. All these things may, however, cease at once; the sensorium may again become entirely or partially clear, only to succumb once again to a new disturbance, after a longer or shorter period.

Stomach and intestinal symptoms.—If no vomiting has taken place at the beginning, it then comes to be observed later; in some rare cases it does not occur. This does not continue during the whole time of the disease, but on an average for five or six days; varies exceedingly in frequency; appears from time to time, as soon as something, no matter how indifferent, has been taken into the stomach. The vomit shows nothing special; if the disturbance continues until the end of the disease, it is possible that slight admixture of blood may be found, as in one case of a girl, of nine years (hemorrhagic ulcers of the mucous membrane of the stomach). The appetite is generally gone (in two cases observed by us the patients ate abundantly, but vomited again immediately); the tongue is coated, but moist; thirst moderate. Constipation may be wanting, if it has been energetically met from the beginning, or if there exist an old affection of the intestines; otherwise this is a very constant symptom. Some cases have been seen in which there was diarrhœa till the end, without ulcerating affection of the mucous membrane.

Convulsions and contractures.—In the case of children there often occur general cramps accompanied by chills; in individual cases, indeed, these dominate all other symptoms. They are, it is true, very various in form and intensity. We shall not here enter upon a more careful description. In the case of adults, epileptiform convulsions are rare, and indicate a form of disease not according to the usual type. Partial convulsions are seen in the case of children and adults: tremor of the eyeballs; twisting of the same, where both eyeballs make the same, or less often dissimilar movements, so that strong transitory convergences and divergences occur; grinning contortions of the muscles of the face; grinding of the teeth; finally, two well-known and very important symptoms begin to show themselves, to disappear, to appear again, and at last to remain, viz., the stiffness of the muscles of the nape of the neck and of the back, and the retraction of the abdominal muscles—belly like a boat.

Sensitiveness.—In regard to the more delicate conditions of sensitiveness at this period, we are really not clear. If the consciousness is not deeply deranged, we arrive at normal or nearly normal results, as soon as we succeed in fixing the attention of the patient; if the psychical derangement increases, there is an end of all accurate examination. From time to time we notice a general hyperæsthesia of the whole surface of the skin, as at the beginning of the disease.

Paralyses in the face may even now occur; there is not, however, the slightest regularity as to the time of their appearance; one pupil may be wider open; ptosis may occur, slight facial paralysis, or strabismus.

Examination of the retina.—Where general miliary tuberculosis exists, it is not uncommon to find tubercles of the choroid; where there exists tuberculosis of the pia mater alone, these are, strangely enough, and for some hitherto quite unexplained reason, very seldom found.

But even at this time there may be seen a marked stasis in the veins going from the papilla. This stands in close connection with the change of character of the fluid coming from the subdural space (arachnoid sac of old writers) into the sheath of the optic nerve (see below).

Derangement of the vision does not seem to be serious, or indeed seems entirely absent, so long as the patients are tolerably conscious.

Pulse and fever.—The pulse may, in the most remarkable manner, in the case of adults, show during many days not the least abnormity beyond slight variations, marching generally parallel with the temperature. But in other cases it is from the beginning somewhat slow—60 to 70, full; it is, moreover, subject to rapid variations; muscular exertion of every sort and slight psychical excitements cause it to rise, especially in the case of children. With these latter we find not very unfrequently an irregular pulse so early as after the first twenty-four to thirty-six hours. The fever shows the greatest inconstancy, so that we must not think of presenting a typical curve. Hyperpyretic temperatures do not occur at this time. There are generally afternoon or evening exacerbations up to 39° C. (102.2° F.), or 39.5° C. (103° F.) ; in the morning the temperature varies between 38° C. (100.5° F.) and 38.5° C. (101.5° F.) ; indeed, in the morning there is often apyrexia. The condition of the skin, too, shows marked differences caused by the preceding diseases and the individuality of the patient. Sweats are generally wanting ; there is, on the contrary, a disposition to slight turgor of the skin and to dryness. The injection of the face and of the conjunctiva is very varying, and indeed, in the case of the same patient, from one hour to another, without this variation being in connection with any discernible incidents. Lividity of the face belongs rather to a later period, or is dependent on complications in the lungs. In some cases an exanthematous roseola was seen on the abdomen ; in one such case under our observation there was general miliary tuberculosis as well as tuberculosis of the pia mater. Herpes labialis is very rare. Quite as rarely is there mention of icterus, the character of which can hardly be discussed, owing to want of results of observation.

The easily occurring and intense redness of portions of the skin subjected to pressure is very frequently observed, but is by no means characteristic.

If now to these symptoms there are joined the signs of a quickly increasing inward pressure, we may consider this to be the beginning of the second stage ; the transition is, however, seldom sudden—generally gradual. Signs of an affection of the base are mingled with the others, without it being possible to discover the slightest regularity in their sequence and arrangement.

Serious psychical lesions come more and more to the foreground. Reaction is slow, uncertain, often entirely absent ; dull brooding passing into heavy sleep ; often a very suddenly occurring deep coma (in the case of children not unfrequently ushered in by a convulsion), which in some cases continues unbroken till the end, in other cases again gives place to short clear intervals. The signs of excited delirium become less frequent ; the symptoms of psychical torpor have more and more the upper hand. This is the time when the hydrocephalic cry, in the case of children, is most frequent; adults, too, in the midst of sopor still give evidence of severe headache by their groans and gestures, indicative of pain.

Cramp symptoms make their appearance, considerably more developed than before; combined sluggish movements of the eyeballs; nystagmus-like spasms of the muscles of the eye; the pupils are often rather unequally open; they are moderately open, and then slightly responsive or entirely irresponsive. In the tract of one or other facial, there occur slight convulsive movements, giving rise to the expression of face of laughing or crying; likewise isolated contractions, of very short duration, of the motors of the fingers and toes, are frequent, but less remarkable. Again, there appear convulsive movements of entire groups of muscles, which repeat movements with an object, as automatic movements of chewing, winking the lids, whistling, chattering with or grinding the teeth, shivering movements of the arms, etc. The stiffness of the muscles of the nape of the neck is more constantly present, and also gains the muscles of the back; it is more intense, so that the head is thrust into the pillow behind it. Attempts to relax these stiffened muscles generally occasion violent pain, which, in spite of the sopor, is made evident by groaning. Finally are to be mentioned convulsive attacks, which are rare with adults, but with children are, in varying intensity, very frequent.

The previously mentioned cramps in the tract of the facial muscles do not by any means allow a positive deduction as to the existence of serious affection of the base; indeed the paralyses of facial muscles which now chiefly come to the foreground render this only exceedingly probable, but not certain. These paralyses are: paralysis or paresis of the *oculo-motor*, in which case either all the fibres may be affected, or only a part of them. From this result divergent strabismus, ptosis, dilatation and fixity of one pupil, either all these together or discrete, and following in time one after the other. In the case of slight inequality of the pupils, we must not immediately conclude that there is paralysis of the oculo-motor on the side of the dilatation.

Trochlear.—We do not know of any isolated paralyses of the trochlear; in some cases paralysis of this nerve may be recognized, combined with that of the oculo-motor.

Paralysis of the *abducens* leads, because of the greater force of the internus, to convergent strabismus, mostly of one side, seldom of both.

Paralysis of the *facial* leads to dropping of one nostril and one corner of the mouth, to a lessened development of the fold between nose and lip of that side, to a sluggishness of movement of the whole tract of innervation of one side (wrinkling of the forehead, closing of the lids, movement of the nostrils, mouth, and chin). It deserves remark that *total* paralyses of the facial have occurred (affection of the base); then, again, such as in no respect differ from the usual form of cerebral paralyses of the facial (oral and nasal branches); finally, paralyses only of the ocular and frontal branches. Apart from this, that in individual cases the paralysis of the facial is really cerebral, a partial affection of the facial at the base may very easily occur.

Hypoglossus.—Obliquity of the tongue (it turns towards the paralyzed side) occurs, but is not so frequent as affection of the facial.

Hemiplegia and hemiparesis; it is very seldom that we find both extremities of the same side paralyzed; on the contrary, paralysis of one arm, of one leg, or merely weakness, is noted not very infrequently. In the case of simple weakness of the extremities, the autopsy often gives no explanation; in the case of entire paralyses, quite well-defined causes are generally found. One case showed paraplegia, without a sufficient cause appearing at the autopsy; and finally there sometimes occurs a considerable diminution of strength in all four extremities without our knowing where to look for the interruption of transmission. One such case showed marked inflammatory affection of the most superficial layers of both pedunculi; but we have sometimes found the same thing without paralysis.

It is very hard to come to a clear understanding of the accompanying lesions of sensibility. We find in general at this period diminution of the same; sometimes we notice its very sudden extinction in one or the other extremity; in one case it became very early extinct in a trigeminus, so that entire anæsthesia was the result. The activity of the cerebral sensitive centres now begins to become extinct, and indeed, as a general rule, this takes place simultaneously in all of them, though regarding these points one is self-evidently unable to speak very accurately; it is plain that at times patients hear and see nothing at all; at other times a dim, dreamy perception exists.

Retina.—By this time there exists in pronounced form stasis in its veins, as well as a change in the appearance of the papilla, which leads us to conclude that there is œdematous swelling of the same. The veins are dilated, and stand out distinctly on the papilla, which is usually reddened to a moderately intense degree.

Sometimes there appear apoplexies of the retina, but seldom very extensiv

Finally, there appear, by no means, it is true, in all cases, symptoms of neuro-retinitis of various degrees of intensity, either in connection with appearances of stasis, or without these latter. The signs of real inflammation are: disappearance of the distinct limit of the papilla; grayish coloring of the same; slight, veil-like, grayish, half-translucent dimness of the parts of the retina which lie close to the papilla.

Symptoms belonging to the digestive apparatus now fall into the background in the majority of cases. Continual vomiting does occur, but is infrequent; the constipation continues. We have already noted certain exceptions of obscure nature. The urine is mostly passed in bed; there is sometimes retention of urine, rendering the use of the catheter necessary.

The change of the pulse, which usually occurs at this time, although sometimes a little earlier, is of the greatest importance; the pulse, with increasing compression of the brain, becomes less frequent, sinks to sixty, or still lower, is at the same time fuller, and often rather jerky. Still a slight excitement suffices to urge it up to one hundred and over, and in this stage it is then frequently somewhat irregular. The temperature, however, no longer corresponds by any means with these leaps of the pulse; at sixty beats the temperature may remain constantly in the neighborhood of 39° (102.2° F.), and only mark 37.5° (99.8° F.)

or 38° (100.5° F.), while the patient's pulse, by the necessary manipulations, has risen to one hundred and ten or one hundred and twenty.

The respiration frequently shows not the slightest departure from the normal. In some cases it is indeed irregular, with adults as well as with children, and sometimes shows, in most exquisite fashion, the character of the respiratory phenomena described by Cheyne-Stokes. There appears also frequently at this period a not very intense bronchitis where the lungs were previously healthy; once occurred marked hypostasis, which made the previously existing resemblance to typhoid fever still more remarkable. In the case of children there are often found broncho-pneumonic foci and circumscribed atelectases, arising from an acute process, in which cases, however, previous affections of the lungs and accompanying miliary tuberculosis of the lungs must be taken into consideration.

In a series of cases (but certainly less than half) enlargement of the spleen can be proved.

With the increase of the intraventricular effusion, the periods of unconsciousness now draw together to one continuous coma; death, however, by no means unfrequently occurs after gradual increase of frequency of the dwindling pulse, without having been preceded by a protracted deep coma. Whoever wishes to hold fast to the division into stages, may count, as beginning at this point, the third period—in the case of adults generally short, in the case of children often very protracted. Moreover, the picture offered during this third period is by no means an entirely clear one; but there very frequently occur vacillations, and from time to time again temporary gleams of responsiveness.

During this coma every spontaneous excitation is entirely absent; no excitation can awaken a responsiveness; the reflex excitability is also gone, in the majority of cases; even on excitation of the conjunctiva and cornea, it is impossible now to obtain reflex action. All the remarkable ("automatic") movements previously noticed cease; the responsiveness, on excitation of the pharynx, appears sluggishly and tardily, and finally ceases entirely; respiration and action of the heart are the only functions to be observed.

It is in the highest degree worthy of remark, and unexplained, that, in spite of this, there has been seen, by entirely trustworthy observers, a reawakening and relatively correct functionment of the sensorium during a short period usually immediately preceding death.

The motor symptoms are about the same as before ; paralyses seem sometimes less well-defined, since the test is wanting of comparison with unparalyzed muscles. Cramp symptoms (trismus, rigidity of the muscles of the back, rigidity of the extremities) are still frequent ; there are, moreover, also present partial tonic spasms of the muscles (contracture of the arm, of the leg). One patient showed a marked contracture in the tract of the facial of one side ; the other was paralyzed. With adults convulsions seldom occur at this time ; with children, on the contrary, general eclamptic spasms do still occur at this stage, and often imply quickly following death ; partial convulsions, convulsive spasms, of one or both extremities, are still seen at this time. The paralyses are likewise very evident ; in regard to this, we may refer to what has been said above. But the isolated paralyses soon give place to a general relaxation of the whole muscular structure. Perceptive susceptibility to excitations of the peripheral ends of the nerves is entirely suspended during the states of deep sopor ; activity of the senses has likewise ceased.

The examination of the eye generally shows the pupils dilated fixedly ; the want of parallelism of the axes of the eyes, existing at a previous stage, is now often not distinctly observable, owing to the entire absence of innervation of all the muscles of the eye ; there is drooping of both eyelids. The retina is likely to show the changes already mentioned in a still more advanced degree ; there are signs of marked stasis and œdema of the papilla with commencement of neuro-retinitis.

In the case of young children the bulging of the fontanelle is now characteristic ; its pulsations become weaker and weaker, and may disappear before death ensues. This shows that the cortex of the brain and the pia mater are already shut off from the circulation of the blood ; this does not reach them any more : these have therefore succumbed to functional death, while the functions of the medulla oblongata still continue.

The pulse shows at this period a decided tendency to become more frequent; it reaches 120, 130, 140, growing at the same time very small and irregular.

The respiration is exceedingly irregular, often forced, deep, snorting, like that of dyspnœa; at other times exceedingly superficial, sluggish, and difficult to detect.

In the course of the disease there occurs rapid emaciation; especially are children reduced to a skeleton before the centres of the medulla oblongata necessary to life succumb to the paralysis, which finally is entire. The skin in these last stages is generally dry and scales off; pallid and without any turgor; with the decreasing force of the heart a slight lividity manifests itself in the face. From time to time a bed-sore appears; in some cases slight œdema, to be ascribed to affections which have previously existed.

During this whole period the spleen continues of the same size which it showed at about the beginning of the disease. According to what has been communicated, the spleen is in a series of cases enlarged; in the remaining cases normal; we do not dare to offer to give figures. The conclusion is not always authorized that in case of enlargement of the spleen this must contain miliary tubercles, since enlarged spleens—in typhoid, for instance—are seen entirely without miliary tubercles.

The temperature curve.—It results from the comparison of a great number of curves that the beginning of the disease does by no means show anything characteristic in regard to the conditions of fever. Moreover, until the very end, the course of the fever is, as we have already remarked, in the highest degree vacillating and irregular. There are cases which, in the second half of the disease (during the stay at the hospital), scarcely show any fever, where temperatures up to 38.5° (101.2° F.) or 38.8° (102° F.) exist for one short period; but with this exception subnormal temperatures are the rule, and these are not influenced even by the agony. In other cases, however, differing anatomically very little from these last, the fever gradually and slowly increases until the approach of death, breaking off immediately before death with a temperature of collapse, or remaining high—differences whose cause remains undiscovered. In still other cases there exist moderate temperatures, remaining the same until death. Finally, cases have been observed—these are, however, not frequent—showing hyperpyretic temperatures attained to by bounds.

Death frequently influences the temperature to a very great degree. There is a sinking of the temperature during the agony, so that 35° (95.° F.) and 34° (93.5° F.) may be reached with the pulse at 130 and 140. There is also, on the other hand, a rise of temperature during the agony; the thermometer marks hyperpyretic temperatures, and continues to rise somewhat even after the moment of death. Chills are very infrequent.

In all cases which have been so examined the electric behavior of the muscles has proved normal to all modes of application of the constant and induction currents.

Urine.—This shows decrease of quantity and increase of specific gravity; in regard to the urea, our observations show divergences difficult to explain; sometimes the quantity of this substance was normal, sometimes increased, and in a third series of cases diminished. The chlorides in like manner show noteworthy variations. Increase of the phosphates is mentioned: we were not able to discover that this was invariable. Albumen is frequently present in small quantity, which varies in the course of the disease; we have never found sugar.

We now turn to a number of cases which had an abnormal course. According to the description of the disease, which has been shortly sketched above, it would seem an easy matter to recognize it; and yet this disease may cause the greatest difficulties to the most practised diagnosticians:

1. There are cases which have a relatively very rapid course, in which evidently a process in the brain—the nature of which is as yet unknown—causes death.

15. A man, nineteen years old, admitted June 16th; his mother had died of tuberculosis; he had had pneumonia four years before, leaving a cough; several attacks of hæmoptysis; admitted to the hospital on account of cough, weakness, increasing emaciation.

17th.—Weak, emaciated, pale, sensorium entirely clear; temperature, 37.6° (100° F.); in both lungs signs of induration and formation of cavities.

21st.—In the morning dull headache, which increases during the day; lies quiet, with pallid face, apathy, eyes cast down; in the afternoon bleeding at the nose; in the evening vomiting of bile. Abdomen drawn in; soft; pupils normally responsive; temperature, 37.6° (100° F.).

June 22d and 23d.—Persistence of headache without intermission; the retraction of the muscles of the belly becomes complete (belly like a boat); from time to time vomiting of the remains of food; indifference; quiet posture on the back, with the thighs drawn up; temperature, 37.7° (100.2° F.)—37.9° (100.6° F.).

June 24th.—Condition of sopor; drawn up in a contracted posture on the right side; when awakened, by being called to, the eyes are slowly opened; the pupils are wide open and sluggish; answers are slow and incomplete; he will show his tongue, but sinks into slumber again as soon as excitation ceases. Facial nerve intact, strabismus convergens. The left upper extremity is more sluggishly moved, and when raised falls back flaccid. Abdomen very much drawn in; constipation.

25th.—No change; sopor and strabismus the chief symptoms.

26th.—Death in the morning. Duration: five days.

Autopsy.—Pia mater slightly œdematous; veins very full; the pia mater very cloudy between the chiasma and pons, likewise in the fissure of Sylvius; miliary tubercles; the upper side of the cerebellum shows decided infiltration. Lateral ventricles *not* enlarged. Substance of the brain flaccid, cortex flaccid, almost diffluent. (Traube.)

There are cases of still shorter duration; there exist without doubt marked differences in the tolerance of individuals for the previously described lesions of the brain, so that here also weakened constitutions offer from the beginning less resistance; we can especially observe this in the case of children who have been previously diseased. The above case of Traube may serve at the same time as a type for cases where ventricular effusion is wanting.

2. Other cases have an exceedingly protracted course; the symptoms are developed slowly; are interrupted; the decline of the intellect is very gradual. Cases are described with a duration of fifty-four, of forty-two days. A remarkable case of a boy of eight years resulted in death on the twenty-ninth day.

3. There are cases in which, in spite of the hydrocephalic effusion, there is not developed a complete *coma*. Consciousness is indeed suspended; the patient does not know where and in what condition he is; he lives a life of the most troubled and confused dreams; but this itself calls up an uncommon excite-

ment—to the very end an unceasing, purposeless, and objectless activity of all the muscles of the whole body. (See a case of Biermer, given in Seitz, Meningitis Tuberculosa, p. 237. The autopsy showed ventricular effusion.) In regard to the cause of departure from the usual type, we can state nothing positive, owing to want of accurate anatomical observations.

4. Individual rare cases begin, in a most remarkable manner, with a sudden paralysis:

16. A man, fifty-two years old; admitted March 31st; comes into the hospital with a complete paralysis of the oculo-motor, which occurred on waking, in perfect health, three days before; the right upper eyelid drops, covering the eyeball completely; impossible for him to raise this eyelid; he can close the lids; right pupil wide open and fixed; movement of the eyeball upwards, inwards, and downwards impossible, but movement outwards in his power. Beyond this no lesion of the nerves. In his mental condition the patient showed strange hasty behavior, restless movements, little attentiveness, sudden quitting the conversation; sensibility good; speech good, though hasty. Patient is weakly, shows evidences of atheroma. Thorax: On the right side in front and below, dullness from the third to the sixth rib, and from the right papillary line to the middle of the sternum. In the same place below there is moderately increased but not distinct bronchial breathing, with rattling half dry râles; no crackling; at the other points vesicular breathing. Heart sounds healthy; spleen not enlarged; no expectoration; no vomiting; no stool. Neither headache nor giddiness.

Four weeks before, patient had headache limited to frontal and temporal regions of the right side. 38.4°–39.6° (101°–103.2° F.); pulse, 96–100. Night of April 1st to 2d.—Restless; patient is delirious.

April 2d.—Hasty manner increased, patient makes trembling, objectless movements; cannot draw on his clothes; senseless talk. Ptosis appears somewhat less. 38.4°–39.6° (101°–103.2° F.); pulse, 96–108. No stool; no vomiting. At night restlessness and delirium.

April 3d.—Same condition; relative clearness alternates with bewilderment· afternoon, attempts at escape; temperature the whole day, 39° (102.4° F.); pulse, 92–100; no complaint of headache.

April 4th.—All the faculties obstructed. Patient speaks constantly with himself, his eyes half open; tremulous movements of the arms, legs, jaws, tongue; clutches about at the air; his arms stretched out before him; tears up his shirt and chews the pieces; his speech is sometimes an indistinct murmur; offers resistance to passive movements, and stiffens himself. Right eye as before; to this is added paralysis of the left abducens; slight facial paresis of the left side; on standing and walking, unsteady motions. Sensibility scarcely ascertainable. Temperature, 37°–37.6° (98.8°–99.8° F.); pulse, 80–92, full; no vomiting; no stool; passes urine in bed.

April 5th.—Same condition; senseless muttering; ceaseless movement of the extremities; makes motions as if blowing tobacco smoke; chewing movements; eyes as formerly, with exception of the left eyeball, which is turned outward. Sluggish responsiveness to strong excitation; stool and urine passed in bed; temperature, 37°-38.8°-40° (98.8°-102° -104° F.); pulse, 100-132.

Death at 8 o'clock in the evening. *Autopsy.*—Pia mater moderately rich in blood; œdema; numerous milky dim places; convolutions not flattened; at the apex of the right gyrus uncinatus, the pia mater is impregnated with extravasated blood; the underlying brain substance is soft; the peripheral substance light-yellow, infiltrated with serous matter; by cutting deeper, we come to a hemorrhagic focus, which has destroyed the walls of the fissure of Sylvius to an extent equal in size to a nutmeg. The pia mater of the fissure of Sylvius shows a grayish-white exudation; on the vessels there are single, very small miliary tubercles. Moderate hydrocephalic effusion, white softening. The right pedunculus entirely riddled with capillary exudations. Right oculo-motor remarkably tender; miliary tubercles in the pia mater along the vessels. (Biermer, in Seitz, p. 147.)

A second case of Traube's (beginning with paralysis of the arm—cause: encephaloid focus in the right hemisphere; miliary tuberculosis of the pia mater, with purulent exudation); a further case of our own observation (beginning with total left facial paralysis—cause: abscess that had broken into the Fallopian canal; caries of the petrous portion; general miliary tuberculosis of the pia mater, with suppuration of the base and slight suppuration of the convexity; tuberculosis of the lungs, and cheesy degeneration of the bronchial glands). It is difficult to form a judgment in cases of this kind; the importance of the symptoms which first occur (paralysis of a facial nerve, hemiplegia) can hardly be correctly rated. The succeeding diffuse affection of the brain may indeed be recognized; the cause of the paralysis, in its closer or wider connection, can only be found under the most favorable circumstances.

5. Some cases begin with aphasia. These are highly interesting, on various grounds. Not many are known.[1]

17. A man, of middle age; disease began in October with coughing, fever, swelling of the cervical glands; admitted to hospital in November. Continuation of the symptoms without hæmoptysis. In the beginning of January great emaciation; pallor; senses and intelligence intact; memory good; excitable condition; sub-

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[1] Some cases were published by Corazza a few years ago.

maxillary glands swollen; severe attacks of coughing; abundant sticky sputa; respiration, 24; slight night sweats; some diarrhœa; pulse more rapid; percussion and auscultation normal.

From the beginning of March till March 24th, was out of the hospital. March 25th.—Less coughing than on admission; lengthened expiration on the apices; no bronchophonia; fever in the evening; no headache. In April, emaciation; weakness, and swelling of the glands constantly increase. First half of May, great decrease of appetite; great weakness; from time to time vomiting, and diarrhœa; constant coughing and fever.

May 22d.—On a sudden a feeling as if the right arm were lifeless (engourdissement); intelligence, senses, and motility normal; headache; no facial symptoms.

23d and 24th.—The same.

May 25th.—*In the evening, on a sudden, aphasia;* fruitless attempts to speak; mind remaining entirely clear; right arm paralyzed; sensibility diminished; no headache; chest symptoms less noticeable; no expectoration; pulse 112.

27th.—Moves the right arm somewhat more freely; aphasia; yet patient can force out in quick succession some words of one syllable. Pulse, 100, regular; temperature normal.

27th.—Patient can, after many attempts, speak out quickly the name of the physician, not his own name; the right arm moves. 28th–31st.—Quiet; somnolence; power to move the right arm the same; its sensibility seems greater; no cough; no expectoration; pulse regular, 90–100. 31st.—Erysipelas of the face; somnolence increased. June 1st.—Somnolence continues; inability to speak continues; cannot name well-known objects. 2d.—Weakness increases; face expressionless; pulse, 100; at night a continuous cry without special character. 3d.—Contracture of right arm; in the evening strabismus; day and night continuous cries. 4th.—Cries less; strabismus continues; pulse, 140. 5th.—Death.

Autopsy.—Subarachnoid œdema; slight injection of the pia mater; three spots on the left hemisphere, caused by slight infiltration of yellow serum in the depression corresponding to the fissure of Sylvius. In the left fissure of Sylvius the pia mater is both grayish and rosy—the former occasioned by a gray, half-translucent mass diffused over convolutions and depressions, and which is sprinkled with gray granulations not larger than a hemp-seed. *In the right fissure of Sylvius there are neither granulations nor gray substance.* In the lateral ventricle half a tablespoonful of clear serum; the walls entirely healthy, as is the whole remaining brain; the cervical lymphatic glands enlarged, and having undergone cheesy degeneration; both lungs filled with a great number of crude tubercles; bronchial glands enlarged and chalky. (Lediberder.)

18. Man, thirty-four years old; hereditary predisposition, phthisis; the affection of the brain begins with chills; severe headache; the pupils and, generally, the motility normal at the beginning.

December 8th.—Restlessness; congestive redness on the head; *in the evening, suddenly, aphasia;* incorrect naming of objects; great restlessness and excitement caused by this; consciousness entirely clear.

December 9th.—Speech quite unintelligible; restlessness; slight signs of right facial paralysis; convulsive movements in the extremities.

December 10th.—Somnolence; aphasia still exists; attack of convulsions. The rest of the course of the disease was remarkable for its severe convulsions.

Autopsy.—Old tuberculosis of the lungs; spleen, kidneys, liver, show miliary tubercles; pia mater much thickened on the convexity, still more so at the base, especially at the chiasma, and most of all in the left fissure of Sylvius; abundant small miliary knots; hydrocephalus; choroid plexus showing tubercles. (Traube.)

19. Man, thirty-five years old; enters the hospital August 28, 1874; mother died of pleuritis; father, cause of death not known. Of their four children, two have died of consumption. Has had no serious illness, although always weakly; never any cough nor hæmoptysis; in his youth onanismus, and since then frequent pollutions. August 4th.—A stitch in the right side. August 11th.—Chill; hotness; severe pains in the right side; severe dry cough; varying improvement; change for the worse; had kept the bed, on which account, at his entrance, there were already threatening bed-sores. August 28th.—No emaciation; muscular system in normal condition, yet anæmic; pityriasis; twenty-six respirations; right side of thorax breathes less fully; rubbing to be felt in the right lateral region; no vibration on speaking; slight dullness under the right clavicle; on the upper edge of the third rib the dullness is complete, and runs into the liver dullness below. On the left side, everything normal; heart normal, somewhat pushed towards the left; behind, on the right side, dullness, beginning at the spinous process of the scapula. In both supra-clavicular hollows the expiration is prolonged; in front, on the right, very slight vesicular breathing; the same in the lateral region; behind, beginning with the middle of the scapula, bronchial breathing and crackling, rubbing; temperature, 38.4°-39° (101° to 102.2° F.).

August 29th.—39.4°-39.8° (103° to 103.8° F.). 30th.—38.8°-39.8° (102° to 103.8° F.); urine without albumen; no nervous symptoms.

Sept. 6th.—Fever in the evening continuous; 38°-39° (100.8° to 102.2° F.).

8th.—Physical examination the same; fever in the evening; 38°-38.8° (100.8° to 102° F.).

12th.—Fever in the evening; physical examination the same; increasing weakness.

16th.—Fever in the evening continues, 37.4°-38.8° F. (99.3°-102° F.) The effusion has decreased; the upper limit is lower than it was.

19th.—Same condition. Over the right apex rather dry râles.

21st.—Fever in the evening, 38°-39° (100.5°-02.3° F.). Effusion diminished; above more vesicular breathing; rubbing sounds.

27th.—Fever continues, in spite of treatment.

October 8th.—Fever in the evening; effusion stationary.

20th.—No change; diarrhœa, which, however, ceases again at once.

November 2d.—Suddenly temperature at 40° (104° F.); darting pains in the right side; rubbing on left lateral region. Coughing with rare catarrhal sputa.

4th.—Fever continues. Slight effusion on left side, below; oppression; want of appetite.

8th.—Same condition; temperature, 38°–39.4° (100.5°–103° F.). Effusion on left side somewhat higher; right, stationary; dullness very marked, but respiratory sounds to be heard downward far beyond the limit of dullness. Diagnosis: Purulent effusion with membranous formation.

18th.—For several days complains of not very intense headache; fever continues; physical condition the same; mental faculties entirely normal.

23d.—There comes suddenly a strange confused behavior; patient does not answer; does not well understand questions; pupils contracted; very little responsive; very slight paresis of the entire region supplied by the right facial.

24th.—Same condition; confusedness; want of clearness; slight delirious attacks at night.

In the evening suddenly aphasia; does not find the right words; takes pains to answer; is vexed when he does not succeed; no further paralyses; there are none of the extremities; gait somewhat uncertain. Temperature, 38.4°–39° (101°–102.3° F.). Pulse, 114–120. No headache; later more confusedness; patient is ill-tempered, abusive, fragments of curses, etc.

Diagnosis: Circumscribed tubercular focus on cortex of left island of Reil.

25th.—Patient's intellect clearer; aphasia less marked, yet there still remain quite a number of sounds which he seems to have forgotten how to produce; has a dim remembrance of what has happened; no headache. Temperature, 37.4°–38.2° (99.4°–100.8° F.). Pulse, 102–96.

26th.—Variability in the intensity of the aphasic lesion. In the evening it is again very intense. Patient can write; writes down correctly a short dictated sentence. Temperature, 37.6°–38.4° (99.8°–101.2° F.). Pulse, 90–102.

27th.—Same condition. Temperature, 37.6°–38.6° (99.8°–100° F.). Pulse, 90–96.

28th.—Speech and sensorium as before; slight facial paresis of the right side; *beyond this no paralysis;* no stiffness of the nape of the neck; no retraction of belly; no vomiting. This latter has been wanting from the very beginning of the brain lesion; constipation; pupils moderately open; slightly responsive, equal; no ptosis; no strabismus. Examination of the lungs gives same result as before. Temperature, 37°–37.6° (98.8°–99.8° F.). Pulse, 84–114.

Evening.—Increased loss of consciousness.

29th.—Entire deprivation of the faculties; no conversation possible; slight delirious attacks; incomprehensible muttering; picking at trifles with his hands; no change in the nervous appearances. Temperature, 37.5°–36.8° (99.6°–98.3° F.). Pulse, 114–132.

Death at four o'clock on the following morning; œdema of the lungs.

Autopsy.—Edges of both lungs fastened by old adhesions; purulent pleuritic effusion of the left side, reaching to the middle of the scapula; thick, skin-like pseudo-membranes, full of vessels, forming into connective tissue, and enclosing the effusion. In the left lung an old cavity, of the size of a hazelnut, with ulcerated walls; in the vicinity, induration of a slaty gray color with small cheesy foci, and very numerous miliary tubercles scattered through the whole lung; lower border compressed; the right lung shows similar changes; the other organs are without miliary tubercles; the glands are free.

The dura mater is easily torn; in the subpial space is a considerable amount of watery fluid; along individual vessels on the temporal and occipital lobes of the left side there is purulent formation in spots and streaks; at the base, around the chiasma, there is a pulpy purulent infiltration of the pia mater, of slight extent. *The right fissure of Sylvius is entirely free from this; on the other hand, it enters the left fissure of Sylvius, and, following its branchings, covers the whole left island of Reil; and following certain vessels, spreads even beyond the operculum; in the pia mater of the left fissure of Sylvius are numerous miliary tubercles;* these are also found in the tract of the first frontal and temporal convolution—of the anterior and posterior central convolution of the left side, but in very moderate number; *in the right fissure of Sylvius, no tubercles;* the right hemisphere, indeed, is entirely free from them; slight hydrocephalus; no softening; in the left island of Reil superficial softening and capillary apoplexies.

As opposed to this case, we now offer one in which the formation of miliary tubercles was confined exclusively to the right fissure of Sylvius:

20. Student, twenty-two years old. Old phthisis, lately much aggravated; night sweats; fever; infiltration of both apices, with consonant rhonchi; elastic fibres in sputa. New illness with nervous symptoms; sometimes absence of consciousness, complaint of headache; slight delirious attacks, giddiness; still everything in very moderate degree, without real exacerbation of fever. This continues forty-eight hours; then the headache becomes more severe; the delirious attacks cease, making room for a stupid indifference and absence of responsiveness. At the end of the third day, there is sudden *paresis of the left arm; the left leg being also weaker than the right;* patient can, however, still walk; slight lesion of left facial; *power of speech entirely retained; no trace of aphasia.* This continues two days; very insignificant fever; pulse, between 90 and 100; no sinking of the pulse; lies quietly, without much responsiveness, yet no coma; from time to time a correct answer, yet no distinct consciousness of time and place. In the last two days patient sinks into deeper sopor, but shows no positive coma; no motor symptoms of the muscles of the eye; no rigidity of the nape of the neck; the paralyses of the left side continue; the paralysis of the arm becomes severest towards the end; retraction of the belly; no stomach symptoms; constipation. Death on the seventh day, with hyperpyretic increase of temperature, and pulse of 130. Severe hydrocephalus was therefore, from the symptoms, to be excluded even during life.

Autopsy.—Old phthisis with fresh miliary tubercles in lungs; miliary tubercles in liver and intestines; about the chiasma a small amount of pulpy gelatinous effusion with pus, which does not pass the anterior edge of the pons; left fissure of Sylvius entirely free; into the right fissure extends the basal exudation even up to the highest portions of the cortex of the island of Reil; the pia mater of the convexity is free from pus and effusion; on the base and in the right fissure of Sylvius are great quantities of fine miliary tubercles, scattered on the right hemisphere in the tract of the first frontal and first temporal convolutions; *on the left side none to be found;* moderate hydrocephalus; no softening.

From these cases there are various things to be learned:

1. A suddenly occurring aphasic lesion may be used to diagnosticate miliary tuberculosis of the pia mater; there must certainly be reasons present which point to the possibility of tuberculosis of the pia mater—that is, manifest etiological reasons. In all cases of aphasia we must think of the disease which now occupies us, as one possible cause.

2. In all these cases the region of the brain which was affected was the cortex of the left island of Reil. The test case, No. 20, shows that the same affection of the right island of Reil caused entirely analogous symptoms, *with the exception of the aphasia.* No matter how skeptical one may be of the truth of the teaching that aphasia always is caused by lesion of the left island of Reil, the force of observations, like the above, must nevertheless be acknowledged.

3. There is also in these cases no other supposition possible than that the miliary tubercles have grown unobserved in the respective localities, or at least that the signs of the same have been masked by the severe symptoms of disease, elsewhere. The symptomatic beginning of the disease of the brain is here also occasioned by the inflammation of the tubercles *in loco.*

4. In almost all these cases there appeared a paresis of the extremities of the opposite side, arm, leg, facial tract. It is plain that the causes of this lie deeper in the brain, and cannot be specified with sufficient certainty. But we may conclude from this that, in cases of effusion in and near the fissures of Sylvius of both sides, the extremities of both sides are really in a *condition of paresis*, a circumstance frequently passing unobserved, owing to want of opportunity of comparison.

5. In all these cases the hydrocephalus was very limited. The affection of the base did not reach the usual degree, and the

miliary tubercles were confined to the tract of the artery of the fissure of Sylvius. That is to say, the choroid plexus was free. The form of disease during life was in conformity with this, namely, the absence of any considerable compression of the brain, as well as the circumstance that the pulse did not sink in the way that it does in cases of great effusion.

6. We have already spoken above of the isolated affection of the artery of the fissure of Sylvius, and of the opinion formed in regard to this, that we have here to do with a multiple embolism of cheesy masses in suspension in the arterial circulation.

7. The disease may begin with *sudden* unconsciousness; this is either a sudden cessation of all consciousness of self, followed by quiet coma, or it may be a most restless delirium, which, however, lasts only a relatively short time, and of which the patient retains absolutely no recollection (Biermer, given in Seitz, Case 31). In one case observed by us it was necessary that the patient should be held by several men for a number of hours, on account of his blind raging and madness; after this followed very quickly conditions of sopor, alternating with slight delirious excitement, and nothing more of an unusual nature during the course of the disease. Every physician of the insane has seen cases in which the trouble begins with conditions of slight excitement.

8. In some cases the primary affection of the disposition is depression of spirits; this may be so characteristic, that without accurate observations of the fever, it would be considered simple, acute melancholy: anxiety, anguish, hallucination of the hearing, self-accusations (evil-doer, murderess), attempts at escape for fear of punishment. This lasts only a short time. In the case observed by me, quiet followed after forty-eight hours, but with it unconsciousness, very soon after ptosis and facial paralysis, and shortly all the signs of tubercular meningitis.

9. Very often the whole runs its course under the appearance of typhoid fever; this mistake is one of the most frequent errors of diagnosis that happen in the case of tubercular meningitis. We must remember, in this connection, how the characteristic symptoms of typhoid may be concealed where the brain symptoms are conspicuous; how under such circumstances an irreg-

ular curve is formed; how, in individual cases of typhoid, the intestinal symptoms fall to the background, so that there may even exist an obstinate constipation during the whole course of the disease; how vomiting occurs under various circumstances; how roseola is wanting, and enlargement of the spleen may be hidden and obscured; how a diffuse affection of the lungs of a typhoid nature may show a condition of the thorax like that in miliary tuberculosis of the lungs; how, finally, the brain symptoms in typhoid may exactly coincide with certain cases of tubercular meningitis. Excitement and delirious attacks, refractoriness and screaming, hallucinations and illusions, occur; general rigidity of the muscles, isolated rigidity of the nape of the neck, convulsive symptoms of various nature, are observed in one case and the other. Facial paralysis may be wanting in meningitis; symptoms of the pupils of less gravity may be present in typhoid and in meningitis. The curve ought to be decisive; but apart from the sudden falls of temperature, which occur in typhoid and which are not every time to be explained by a particular incident, a short meningitis curve may very easily resemble a typhoid curve. Roseola speaks in favor of typhoid, but is often only partially developed; and an exanthematous roseola has also been observed in some cases of tubercular meningitis. Not even the lesions, observed in the retina, are decisive, *since in the case of severe brain affection in typhoid there occurs a certain degree of venous stasis in the retina*, which does not, however, attain neuro-retinitis. On what this last fact rests cannot yet be clearly demonstrated; the changes in the cortex in typhoid have been up to this time too little examined into; it may be that we have to do with a swelling of the cortex which causes the fluid of the subdural space to be crowded out of its place. We avoid giving cases, such as must have come under the observation of every practitioner. (See cases of Biermer, given in Seitz, p. 358.)

10. Tubercular meningitis — especially in children have we observed exquisite cases of this sort—may follow its course with the appearance of constant compression of the brain, with few mental symptoms in the beginning of the disease. One case, of a boy of eleven years, showed at first slight fever, at night rest-

lessness, the next morning somewhat delirious speech, and only two hours afterwards sinking into sopor, from which, until death, the patient did not for one moment awake; no convulsions; no contractures; all the muscles relaxed; the pupils at first contracted, very soon after wide open and immovable; no spasmodic symptoms; no paralytic symptoms involving the muscles of the eye; dropping of the lids; loss of power over the muscles of expression (*Mimik*); inaction of the muscles of bladder and rectum; nothing noticeable in respiration or action of the heart; responsiveness to excitation of pharynx, and some slight movements of the bowels. The hydrocephalic effusion was enormous, the number of miliary tubercles very small, the effusion at the base scarcely noticeable. Similar cases of Biermer.

11. We have already spoken above of the frequent occurrence of eclamptic convulsions in childhood; we repeat again that these may occur in every stage of the disease; that they often mark turning-points in its course, as death or an aggravation of all the symptoms very quickly follows them; so that the disease makes progress by starts. But even with adults these occur not very infrequently; here, too, they keep the form of eclamptic attacks with slight departures from the type; the convulsions generally begin with the muscles of the head or of respiration. In regard to the occurrence of partial convulsions, all that is necessary has been said above.

12. It is especially in children that we have the opportunity of observing cases in which contractures stand in the foreground; cases in point, of adults, are mentioned by Traube, who considers the contractures to have been occasioned by accompanying encephalitis.

13. There are some remarkable cases (Biermer, Gerhardt), in which, every time that the patient sat up, there occurred attacks of tetanic rigidity. A rational explanation of this phenomenon would be exceedingly difficult.

14. Old affections of the brain may cause the form of disease to vary beyond measure. When tubercular meningitis complicates an old affection of the brain, which has its own series of symptoms, the disease then continues to offer a form which is by no means clear. If we are not quite sure what the primary

affection is, it will be hard to form any opinion beyond the supposition of a terminal meningitis of unknown cause. But that the results of long-existing affection of the brain may have the strongest resemblance to tubercular meningitis is proved by the following case:

21. Man, thirty-nine years old. No history to be had. Patient lies quiet in bed, groaning somewhat from time to time, staring without interest into vacancy, taking no care for anything, not speaking; holds the extremities entirely still, with the exception of slight movements of defence against imaginary attacks. On being loudly called to, patient is somewhat attentive, and even answers; but his speech is muttering and indistinct, so that it is only possible partly to guess at his meaning. The right corner of the mouth lower than the left; right nostril the same; in speaking, the whole right side of the facial tract refuses to act. Pupils equal, moderately wide open; good responsiveness; muscles of the eye act freely. Tongue dry, turns somewhat towards the right. Head falls somewhat backward, yet there is no real rigidity of the nape of the neck, no pain there; patient can stand and walk with slight help, but totters like a bad case of typhoid. We may see, on his moving them, that the right arm, in comparison with the left, is plainly more sluggish; the pressure of the left hand is markedly stronger.

Emaciation; panniculus entirely gone. Skin dry; on the body and upper arm miliaria crystallina; spleen not enlarged; snorting respiration, 40. Abdomen drawn in like a boat. Heart sounds normal. Percussion of the thorax furnishes no symptoms; above, the respiration is louder and coarser on the right side than on the left; no râles, except on the border of right lung below and behind. Yesterday evening, temperature, 38.5–40° (101.4°–104.° F.); pulse, 128–160. To-day, temperature, 39–39.6° (102.2°–103.3° F.); pulse, 140–168.

On the following day the temperature still higher—40.2° (104.4° F.); pulse, 168. Still greater absence of consciousness; almost complete want of responsiveness. Slight spasms in all four extremities; paresis of the facial and hypoglossus as yesterday. Combined convulsive drawing of the eyeballs towards the left; from time to time movements, as in nystagmus, of the two eyeballs; no strabismus. Oculomotor acts freely; pupils moderately wide open, equal; responsiveness exceedingly weak. The right side of the body weaker than the left. In regard to the sensibility we can make no statement.

The examination of the retina shows the limits of the optic nerve somewhat indistinct; veins distended; no tubercles of the choroid.

Very restless in the night; trembling of hands and feet; uncontrolled complex movements; spinning about; creeping from bed, etc.

At one o'clock, temperature of 41° (105.8° F.); pulse, 132; at the close the left pupil opened as wide as the right, which was treated with atropine. Death at half-past one o'clock.

Autopsy.—No effusion at the base; both fissures of Sylvius free; vessels of the surface of the brain entirely normal; œdema of the pia mater; no suppuration or exudation observable anywhere; atheroma of the arteries of the base. Ventricles not dilated; no hydrocephalic softening. In the white substance of the left hemisphere a number of necrotic foci—as many as eight—from the size of a pea to that of a bean. Some similar ones in the right hemisphere.

No trace of miliary tubercles.

Finally, the following case shows how meningeal tuberculosis may be added as final process to a long-existing affection of the brain, of which it is a sequence:

22. Girl, thirteen years old; has been suffering for two years with a disease of the brain; scrofulous antecedents; cicatrices on the neck; has been coughing for some time past; her general appearance is said not to be worse; nutrition has not suffered.

For two years past has had a series of epileptic attacks, which have always been considered as true epilepsy; about every four or five weeks an attack without aura; attacks varying in duration and intensity; long sopor. Has been much treated with bromine. During about six months after the beginning of the attacks there were no other symptoms beyond slight headache. But since that time the headache has increased in a great degree: it comes in the form of severe attacks, lasts a few hours, and ends each time with severe vomiting. No anomalies in the stool. For about one year there has been observed a decrease of memory, of the thinking powers, and of power of judgment. For about one year past the right arm is said to be weaker than the left.

Examination shows a low grade of intelligence, especially a bad memory. Pupil and muscles of the eye, facial, and hypoglossus normal; right arm and right leg weaker than the left extremities, but only in a trifling degree; in their sensibility no difference to be proved; muscular sense retained; speech not hindered; slight interference with the gait, owing to the weakness of the right leg. From time to time the attacks of headache are repeated; the respiration is more hurried during these attacks; the pupils dilated; conjunctiva and face injected. In the course of six weeks there were two epileptiform attacks, *both times with rise and subsequent sudden fall of the temperature.* After the attacks of headache, vomiting. Retina normal; no apoplexies.

The child is taken ill with a final affection of the brain, beginning acutely and lasting seven days. There had been more headache for about three weeks, when suddenly appeared slight fever; constant headache; disordered ideas, want of consciousness, in varying degrees. From time to time slight delirious attacks, from which she may be awakened by calling; then there is sudden loud shrieking; then for hours together she lies in a state of sopor, with lucid intervals, when she complains of rushing noises in the head, of ringing in the ears, of exaggerated headache. From time to time vomiting, constipation. Right pupil is now more dilated and less responsive; no lesion of the muscles of the eye; the old lesion on the right side of the body. On the second day appears slight stiffness of the nape of the neck, which soon disappears again, and shows itself from time to time afterwards; abdomen not retracted. On the third day more sopor and another strong convulsion. From the third day on almost complete coma. On the fourth day both pupils became somewhat more dilated and less responsive. On the fifth day sopor, without new appearances. On the sixth still another slight convulsion, occurring during a state of total unconsciousness. Death in the beginning of the seventh day. The pulse had showed no depression; during the seven days patient was never entirely free from fever, but the fever was moderate and entirely irregular.

Autopsy.—Cheesy focus, of the size of a nut, in the occipital cortex of the left hemisphere; softening near the same. Over the right hemisphere the pia mater simply dimmed; above the left hemisphere this dimness was more decided; pus in streaks along certain vessels; this dimness (of the pia mater) extends on the left side from the occipital point quite to the anterior limit of the anterior central convolution; it extends downwards on the median surface to the corpus callosum and the corpora quadrigemina. No effusion at the base; hydrocephalus without softening; slight granulation of the wall of the ventricle, proving chronic hydrocephalus. Miliary tubercles in the tract of the inflamed pia mater on the left hemisphere; on the right none; in the plexus none; on the base and in the fissures of Sylvius none: all other organs free from them.

There is no doubt that the cheesy focus in the occipital lobe furnished the tubercles which were observed in its neighborhood.

In speaking of this case, we take the opportunity again to call attention to this: that various ways appear to exist in which the pia mater may be infected with miliary tubercles. In the above case there can be no question of arteries and embolic processes, since the beginnings of all the arteries of the brain, coming from the circle of Willis, were free; moreover, the miliary tubercles were placed by no means in the tract of division of one artery alone; finally, no primary focus was found in the body. Exactly the contrary was found in the cases previously cited in regard to the embolic process. But here the tubercles are only on the surface of the brain; they are found in the immediate neighborhood of the old cheesy focus; and here we claim that the propagation takes place through the lymphatic vessels. A similar case of our own observation has less force of proof (old tuberculosis; caries of the petrous bone; cheesy focus between dura and pia mater, joining the two; at that point the pia mater and brain grown together; and cheesy masses even in the cortex; exudation at the base and hydrocephalus; general miliary tuberculosis of the pia mater. The point of departure of the miliary tubercles is in this case not clear).

The conclusions in regard to the origin of disease which can be founded on these symptoms are, we are sorry to say, only partial. In order to form a sure judgment, it would be necessary to undertake in each case a minute examination of the whole central nerve system, and this demand has not yet been complied with.

We have noticed above the fact that, especially in the case of children, a remarkable prodromal period ushers in the disease, a period which is apt to be considered as the time of the development of the miliary tubercles. But just as often does the disease begin, especially in the case of adults, in a time entirely free from nerve symptoms. Thus the individuality plays an important part in the resistance to the noxious influence. As is often the case otherwise, so it is here, too, that the brain of the child suffers most, but in what a remarkable manner! Loss of the activity of the mental processes, intellectual sluggishness and torpor, aversion, disarray of the ideas, forgetfulness, ill-humor, entirely changed relations towards the outer world, analogous to certain psychical lesions of adults; seldom severe headache, but often feelings of giddiness, wandering of the wits; no wildness of the mental processes. The connection of these things with the sprouting of the miliary tubercles is by no means clear. We have really no other choice than to discard lesions of the circulation (Bastian). It may be that epithelial granulations in the perivascular sheaths of the vessels are capable of destroying those constituents of the blood, destined to nutrition, before the brain receives them. The flow of the nutritious fluids may in some way be hemmed in the tissues by the multiple new-formations. Finally, by the stoppage of many blood-passages, individual arteries of the brain may come to lose their normal quantity of blood, which is positively necessary to the fulfilment of the functions. These are all hypotheses, and should only be rated as such. Little by little, however, the growing miliary tubercle comes to be a foreign body, which now for the first time is able to act as a mechanical cause of symptoms of irritation, at the place of its implantation. Hence come, in the midst of the prodromal period, individual symptoms of irritation, slight spasms in children, contraction of the pupils, changing, however, every moment; from time to time a sudden eclamptic convulsion. The countless number of these irritations must be considered as the final cause of inflammation. General fluxion to the pia and to the cortex makes its appearance; to this are added solutions of continuity of the vessels, caused by the tubercle in the first instance, by the fluxion secondarily. These lead to mechanical lesions in the cortex; and, finally, there is joined to this the extravasation of the formed elements of the blood. It is plain to see how exceedingly crude these notions are, and how the most important previous hypotheses are wanting to enable us to follow the individual parts of the process in their strict logical development. But the likeness is here a very close one with the brain affections of typhoid, with certain cases of paralytic dementia, with erysipelas of the face. We recognize in these diseases similar changes of the cortex—only, it is true, by poor scattered scraps of knowledge; but it is instructive to see how the brain disorder of typhoid and the foregoing may coincide in their symptoms, so that, where other circumstances are not entirely favorable, the diagnosis may become an impossibility.

The psychical symptoms.—These, to speak in a general way, go through a certain round; at first excitement and a series of abnormal conditions of mental irritation; then a very variable mingling of the latter with symptoms of torpor; finally, these latter prevail, and are lords of the situation until death. The reason of the condition of excitement seems now to be before us; this may well be occasioned in the cases in point by analogous changes in the cortex having a parallel march with the inflammatory lesions of the pia mater. These changes of the cortex are found, moreover, in the most various degrees of development, and accordingly the psychical lesions are sometimes present, sometimes only foreshadowed. Moreover, owing to the very trifling amount of material collected up to the present time, it is not yet possible to draw a strict parallel between the initial mental manifestations and the changes in the cortex which are anatomically demonstrable.

It is, finally, entirely obscure how the functional anomaly results from the proved changes in the cortex. The origin of hallucinations is without doubt to be ascribed to the excitation of certain nerve-centres of the cortex of the cerebrum; this is not the place to inquire whether it be possible to find out anything about the exact locality of the same. Varying delirious attacks are then the natural results of continued delusions of the senses; and we cannot be surprised that unnatural tendencies and behavior follow according to the laws of psychical causality. Disorders of the mind in a particular direction (disposition to melancholy and mania) are here quite as obscure and inexplicable as they generally are. The remarkable movements of patients (wiping off things, defending themselves, catching at objects, etc.) often lasting for hours together, and showing the character of doing things with a purpose—these are in part results of the hallucinations; in part, if we think that we may exclude these latter, they are occasioned by the occurrence of irritations of a centre of co-ordination lying further within the cortical substance.

Hydrocephalus.—The plexus—some of them affected with miliary tubercles, some of them free from these—come to be inflamed conjointly with the pia mater. The activity of the process in both cases is, however, clearly not always the same; and thus the effusion into the cavities of the brain may be delayed, while the pia mater of the base and the convexity goes rapidly through with its changes; the effusion may in other cases be poured out with unwonted rapidity, and in great quantity; it may, finally, be almost entirely wanting—this where the plexus are either entirely free from lesions, or where the changes in them are of the most trifling description. In any case, the effusion is the cause of those symptoms of compression of the brain which occur at various times, as shown above, and which frequently fall into the background; from this we may gain a good help for diagnosis.

Motility.—It is exceedingly difficult to give, for the lesions of the motility, explanations for the correctness of which we should be willing to hold ourselves responsible.

The various irregularities of gait are in part occasioned by the giddiness which exists; the patient feels as if he were flying or falling; as if the floor went away from under his feet; as if everything turned round with him (as a patient, who long retained his consciousness, well described it). In other cases the difficulties of gait are occasioned by a general weakness of the extremities, which may occur very early; or, finally, by a weakness of the lower extremities, occurring before that of the upper extremities, and in regard to which we do not dare to give any opinion so long as our knowledge of the condition of the spinal column is so incomplete. Once we even saw the disease begin with a weakness of the legs, in near connection with a paraplegia, without being able to come to a definite conclusion as to the cause.

The changes in the speech.—Cases have been mentioned above in which aphasia stood in evident connection with changes in the cortex of the left island of Reil and its neighborhood; the test case with exactly similar affections of the right side showed no aphasia. Thus here again two things seem to be proved: the cortex of the left island of Reil has more to do with the speech than other portions of the cortex; moreover, embolism (*sit venia verbo*) is more frequent on the left side than on the right. We refrain here from an enumeration of the various attempts at explanation.

Moreover, there frequently occur slighter or more noticeable difficulties in the articulation of words. These may be occasioned by lessened conducting power in those nerves which serve to move the muscles of speech (affection of the pia mater of the base); and here the relative infrequency of paralyses of the hypoglossus would be no objection; often, indeed, the innervation of the muscles of *both* sides is exceedingly feeble. Difficulties of articulation may, however, also take their rise in the medulla oblongata; but, as we have no information whatever in regard to this in tubercular meningitis, we can say nothing further.

Rigidity of the nape of the neck.—We do not yet know positively which system of fibres it is the irritation of which causes rigidity of the nape of the neck and of the muscles of the back. Formerly considered as a symptom which pointed to a lesion at the base, this was afterwards seen where there were lesions of the ventricles, and, finally, where there were lesions of the surface; so that one was forced to the conclusion that this (the rigidity of the nape of the neck) had its origin in a third lesion, to which sufficient weight had not yet been given. This was, perhaps, to be looked for in the simultaneous affection of the membranes of the medulla oblongata and of the spinal marrow. This has certainly, up to this time, been very often overlooked. It is by no means always present; but neither is the rigidity of the nape of the neck.

Retraction of the muscles of the abdomen.—The physiological mode of origin of this is unknown.

Convulsions occur at every period, during the inflammation of the cortex and at the period of its compression. We see them reach all possible forms and degrees—partial, total, from slight tremor to protracted eclamptic attacks.

The occurrence of slight, isolated tremulous cramps is probably to be ascribed to the irritation of ganglionic apparatus. In support of the opinion that they proceed from irritation of the centres of Hitzig in the cortex, we can only bring up the reasoning deduced from patho-anatomical examination. To explain the occurrence of more serious convulsions, we must have recourse to the medulla oblongata; we are entirely authorized to do this, on account of the complete conformity of certain ones of such attacks with epileptic attacks. Thus we believe that anæmia or stasis in the medulla oblongata may lead to the eclamptic attacks. We cannot say positively how the former (slighter attacks) are to be explained—whether we have to do with irritation of the vaso-motors, or perhaps with a displacement of blood caused by the pressure from above, or with some unknown hindrance of the circulation.

The *paralyses*, too, the explanation of which seems an easy matter, are not thoroughly understood. The individual paralyses in the face are charged, with more or less reason, to affection of the base.

Oculo-motor.—In a series of cases, its neurilemma on the base was involved in the inflammation; but its fibres were intact. At the same time the aqueduct of Sylvius, on the floor of which lies the ganglion of the oculo-motor, appeared decidedly dilated, the corpora quadrigemina in the same condition, the cortex of the cerebrum as well. Now, in such cases where does the cause of paralysis lie? In the ganglion? In the peripheral conducting part? If the paralysis is total, and there appears dilatation of the aqueduct of Sylvius, we may venture the opinion that the paralysis takes its origin in the ganglion, especially if the course of the nerve on the base is intact. A slight shortcoming in the functions of a nerve, or only partial paresis, is rather to be ascribed to a partial diseased condition of the nerve at the base—that is to say, extravasation of blood about the nerve, thick sheath of pus around it and in between its fibres.

Abducens.—With paralysis of the abducens, we always found a cause at the base, similar to that already mentioned in the case of the oculo-motor. We do not yet know of a central paralysis of this nerve. The ganglion of the abducens lies, too, less exposed than that of the oculo-motor, on the bottom of the fourth ventricle, yet protected from immediate pressure from above by the bend of the facial.

Facial.—Paralysis of this nerve is of frequent occurrence. This is only exceptionally total—for the most part only pronounced in the oral and nasal divisions. In some cases the paralysis has been qualified as *central*, where only a slight sluggishness was noticeable in voluntary movements, and no diminished activity was to be observed in the reflex action. In one case the paralysis was total, occasioned by the breaking through of a small abscess into the aqueduct of Fallopius. The paralysis is, however, mostly partial, and here voluntary and reflex movements have been affected in an entirely equal degree. The cause is found, in the majority of cases, in diseased condition of the trunk of the nerve, occasioned by inflammation of the pia mater, which, moving downwards, has passed even beyond the posterior limit of the pons.

In some cases, it is true, nothing was to be proved on the trunk of the facial and neighboring acusticus, and then nothing remained but to charge the lesion to a hydrocephalic effusion which filled the fourth ventricle. In fact, the hypothesis is a natural one that the bend of the facial nerve in the medulla oblongata, which is so exposed to pressure, may suffer in such cases; but this hypothesis awaits verification. We have never seen a lesion of the ganglion of the facial, situated, as it is, deep within the medulla oblongata, in a most protected place.

Hypoglossus.—Paresis of this on one side is common even in the beginning of the disease; in the later stages a paresis of both sides is frequent; finally, there is temporary complete cessation of this and reappearance of innervation. The cause of paresis of the hypoglossus remains unexplained for me. In a very long series of cases the peripheral trunk was proved to be entirely unchanged in its fibres, even close up to the medulla oblongata. One might feel disposed here again to charge the lesion to the hydrocephalus of the fourth ventricle; but close against the ganglion of the hypoglossus lies the ganglion of the vagus, if possible still more exposed than the former, and its functions did not suffer, or only much later. Further anatomical examination must clear up this point.

Paralysis of individual extremities.—There are early and late paralyses. The early paralyses are always occasioned by some special process in the brain (encephalitic focus, apoplexy, etc.). In the case of late paralyses it is often impossible to trace the matter, since the cause (especially if the spinal marrow is involved) may lie anywhere all the way from the cortex down to where the nerves in question leave the spinal marrow. Every individual case, therefore, requires a very accurate and long-continued anatomical examination. As causes of late paralyses of one extremity we know: softened focus in a pedunculus; group of capillary apoplexies in the capsula interna—a meagre result.

Hemiparesis.—The same is true of this as of the paralyses of single members. Where it appears early, an easily recognizable cause—at the autopsy—is sure to show itself; where it appears later, the post-mortem examination is often without result. One is disposed, whenever there is distention of the ventricle of the opposite side, to give this as the cause; but this does not hold in every case. We are led to think of considerable basal lesion of the pedunculus, especially if there should be oculo-motor paralysis of the opposite side from that of the extremity affected by paresis; but neither does this hold in every case. We are therefore forced to put off for the time a positive explanation of this symptom. In general we may say that the paralyses which occur at an early stage much more generally show a visible cause than such as occur later.

The alterations of the retina and of the optic papilla have not yet come to be clearly understood.

Various pathological appearances have been seen in the region of the papilla in case of meningeal disease (these remarks may serve at the same time for the forms of simple purulent meningitis, to be described further on).

1. *The venous stasis.*—A disproportion exists between the capacity of the arteries and that of the veins: the former are narrow and indistinctly seen; the latter are dilated and winding. We recognize a heaping up of the veins on the papilla, which is somewhat raised; it is at the same time usually somewhat reddened.

2. *Apoplexies* of varying extent.

3. *A commencement of neuro-retinitis descendens.*—For this it is not necessary that the alterations of capacity of the vessels should have reached a very high degree; it is not positively necessary that œdema of the papilla should have gone before; but this, it is true, is often the case. The papilla shows a turbid redness, which makes its outlines indistinct and may cause them almost to disappear. The parts of the retina lying in the immediate neighborhood are also turbid, so that a delicate gray veil is disposed about the papilla and does away with its outlines.

4. *Stasis and œdema of the papilla accompanying the appearances of neuro-retinitis descendens*—a usual form.

It must be that causes of two sorts can be instrumental in the production of these forms, which may occur separately, and again frequently coincide. *On the one hand, the flow of blood from the retina must often be hindered, and then everything is at a stand-still; the conditions necessary to neuro-retinitis do not exist. On the other hand, these conditions must be able to exist without an actual stasis occurring.* Finally, circumstances must exist *which engender stasis as well as inflammation.*

a. Uncomplicated stasis.—The simple statement, that on increase of the intracranial pressure the emptying of the blood into the sinus cavernosus is hindered (Türck, Graefe), has never been satisfactory, namely, on account of the anatomical conditions of the sinus cavernosus and the ophthalmic vein. This opinion was, however, rendered entirely untenable by Seseman's proof, that the ophthalmic vein also empties freely into the facial vein; and that, consequently, stasis in the retina could by no means be caused by compression of the sinus cavernosus—even supposing this to be possible. There then came, in place of the statement of Türck and Graefe, the information, acquired by Schwalbe, that the space between the optic nerve and its sheaths communicates with the arachnoid space. In spite of this important advance, the venous stasis was not explained, the anatomical conditions were not exhausted. With the optic nerve there advances into the interior of the orbit not only a prolongation of the dura mater, but a prolongation of the pia mater as well, of its outermost somewhat thickened layers—the arachnoid of old writers. The optic nerve, at its entrance to the sclerotic, is therefore surrounded by two sheaths:

1. By the pia mater, which leaves between itself and the optic nerve a delicate network—the prolongation of the space formerly termed subarachnoid, now known as *subpial space.*

2. By the dura mater; between this and the pia mater (that is to say, its outermost thickened layer, formerly termed arachnoid) there lies a second space like a chink—the continuation of the so-called arachnoid sac, now *subdural space.*

With this the anatomical foundations are laid for comprehension of what follows, if we call attention once more to a citation of Hitzig, already mentioned, *that during life, under normal circumstances, the subdural space contains cerebro-spinal fluid.* With animals this is even present in considerable quantity, and disappears quickly after death.

Let us now consider the following cases, which are not unfrequently observed.

I. Meningeal tuberculosis *without* exudation at the base, but with hydrocephalus; simple hydrocephalus of children without miliary tuberculosis; further, every process, causing pressure from within, that increases the volume of the brain (tumor, abscess, chronic hydrocephalus). *All these conditions will occasion a pressing out of the fluid from the subdural space;* this fluid escapes by all the ways which stand open to it; a quickly increasing pressure will likewise very readily introduce a small portion of it into the subdural space about the optic nerve. By this means a sort of mechanical strangulation of the optic nerve, at its point of entrance into the sclerotic (foramen scleræ), is occasioned (formation of the ampulla), and the result will be a hindrance of the flow of blood from the retina, dilated veins, œdema of the papilla, swelling of the same, and heaping up of the veins. Here, therefore, there is no question of any inflammatory lesion, and this agrees with much that is observed during life; we must not, however, infer from the absence of neuro-retinitis that there is absence of exudation at the base!

In simple hydrocephalus (leptomeningitis) of children the stasis is greater than in tubercular meningitis (Horner).

II. Meningeal tuberculosis with exudation at the base and widespread suppuration in the pia mater, but slight or no hydrocephalus; or simple basal meningitis (see following section) without tubercles; or widespread meningitis of the convexity which involves the base as well, *e. g.*, in caries of the petrous bone. *A venous stasis in the retina does not occur*, because the intracerebral pressure is not here so greatly increased, let the lesions of the cortical tissues and the psychical lesions occasioned by these be as marked as they may. But the possibility exists, and often comes to be a reality, that setting out from the base of the brain, the inflammatory lesion may force itself along the course of the optic nerve through the subpial space, reach its point of entrance and advance to the tract of the papilla and to the retina. This would be the uncomplicated neuro-retinitis descendens of meningeal affections (no formation of bulgings of the outer sheath of the optic nerve). In consonance with this opinion is the fact that we have in many cases found a fluid, rich in cells, under the sheath of the optic nerve, on slightly lifting this away. Unfortunately, at the time when these cases were examined, the subdural and subpial spaces of the optic nerve were not distinguished apart. In the case of simple bulging of the outer sheath of the opticus, the fluid is without cells.

III. Tubercular meningitis, with exudation at the base, and hydrocephalus. In such a case the circumstances mentioned under 1 and 2 will be combined. We find on the retina the signs of both changes, usually those of stasis at first and then those of neuro-retinitis. That this form should be of more frequent occurrence than the second, comes from the very nature of the disease, since the absence of hydrocephalus is less common than its presence; this form is, however, less frequent than the uncomplicated stasis mentioned under I. This latter, from the nature of the case, occurs early, while neuro-retinitis requires more time for its development than does a simple abnormity in the distribution of the blood, caused by a continually fluctuating pressure.

All that is necessary has been said above in regard to chorioideal tubercles; we again call attention to the fact of their rare occurrence in tuberculosis of the pia mater.

Diagnosis.

Experience in the case of tubercular meningitis teaches, what indeed we learn every day in the diseases of the brain, that there is for this no pathognomonic symptom or lesion (except perhaps tubercles of the choroid, and these point more to general miliary tuberculosis), but that the diagnosis is founded much more on the *grouping of the individual factors of the disease before us, on the succession of their development, on the basis on which the disease arises.* A normal case following the typical course offers no difficulties of diagnosis, since special symptoms may be easily followed and pointed out in various directions. Without these an inflammatory lesion of great intensity in the brain may be recognized; in case of progressively developed signs of an inward pressure, we should be forced to think of that inflammatory affection which, according to our experience, most often leads to hydrocephalus in the acute manner, previously described; finally, led by experience, we easily recognize a number of conditions in which the disease is developed (scrofulous antecedents, a proof of tuberculosis of the glands, tuberculosis of the lungs, etc.). Difficulties which cannot be resolved are often caused by the abnormal cases, of which a series has been described above, and to which we might have added quite a number showing less marked abnormal variations in their course. We only call attention to this, that in individual cases most unusual symptoms occur in the very beginning (sudden initial paralysis of the extremities, aphasia), which might very readily lead us away from the true direction ; that in cases of slight hydrocephalus the depressions of the pulse, which are so characteristic, may be absent, as well as the comatose conditions ; and in regard to the anomalies of constitution, *so desirable for the diagnosis*, these are not always apparent. Persons to all appearance in the most flourishing good health, have the enemy with them in the form of cheesy degeneration of the bronchial glands, and succumb to this ; so that externally visible traces of scrofula are by no means indispensable to the diagnosis of tubercular meningitis. If we pay attention to these facts : that in one case very many miliary tubercles are present ; in another very few ; that in one case these have all been set and have grown together ; in another case scattered and successively ; that, in consequence of these differences, the individual cases *must* radically differ,—if we note all this, it will be clear to us, that, even with more thorough information, mistakes of diagnosis will still be repeated.

1. In those cases in which an error of diagnosis is impossible, owing to the clearness of the symptoms, may we in those cases more accurately designate the development of the individual parts of the process going on in the brain?

If a decided pressure of the brain does *not* occur during the whole course (see the cases described above of the limitation of the miliary tubercles to the tract of a single artery), if a pulsus cephalicus is not observed, if no symptoms of stasis in the retina are developed, but perhaps signs of neuro-retinitis, then the conclusion is a fair one, proving just in the majority of cases, that the inflammatory affection of the plexus is less developed, consequently the hydrocephalus moderate or exceedingly slight.

This conclusion has not proved correct in individual cases, which for the time remain obscure, like many others. We must be careful not to wish to exclude the affection of the base from the circumstances mentioned above.

If the psychical lesion is very serious—for instance, if very acute and noisy delirious attacks continue, without ending in states of coma—we may conclude that there is intense inflammatory affection of the pia mater and the cortex. In such cases a purulent condition of the convexity would seldom be wanting, and the alterations in the cortex, on account of their intensity, would be readily found.

A facial paresis does not by any means authorize the conclusion (according to what has been previously stated) that there is serious affection of the base. On the other hand, this is indicated by a total paralysis of the oculo-motor nerve in all its branches; experience teaches, that at the base, or in the oculomotor nerve, in the great majority of cases, some quite special lesion has occurred (hemorrhage, infiltration of pus, compression, etc.).

Total paralysis of the facial nerve in all its branches may be taken in all security as a symptom of lesion at the base, or in the course of the nerve in the aqueduct of Fallopius. If the paralysis occurs at the beginning of the disease, the latter hypothesis is the more probable; the occurrence of this paralysis in the later stages is very infrequent.

Combinations of various paralyses in the face are a surer sign of an affection at the base, especially if they are observed crossing, the abducens of one side, and the facial of the other, etc.

Stasis in the retina alone, indicates only general swelling of the whole brain; positive neuro-retinitis points to an inflammatory affection at the base. This refers only to the foregoing acute conditions; in the case of chronic affections of the brain this statement would have to be modified. (See Abscess of the Brain.)

In regard to the quantity of miliary tubercles in the pia mater, no judgment can be formed.

2. Some specially remarkable symptoms may supervene where the type of disease in other respects corresponds with that

of the disease in question, and these may seem to give the matter quite another form; these should be kept in sight, but the diagnosis, if good grounds for it exist, should not be abandoned; such occurrences are:

Suddenly occurring paralysis of a muscular tract, in the beginning or in the course of the disease.

Aphasia: this points to the artery of the fissure of Sylvius, and the diagnosis is hereby only strengthened, where other circumstances are favorable (a plain etiology).

Uncomplicated severe compression, dominating everything else, and allowing no other nerve symptoms to appear, does by no means exclude the idea of miliary tuberculosis. This may be developed with the greatest rapidity, so that the initial period of irritation escapes notice.

Brain symptoms may already have existed before the appearance of such as are characteristic of tubercular meningitis. Since the diversity of these pre-existing affections of the brain is not little, it is hard to give rules of diagnosis on this point. Cheesy foci in the brain cause symptoms which indicate a tumor, but the resulting meningitis will still remain unknown in the majority of cases. The last stages of an affection of the brain (multiple necrosis, abscess of the brain) may personate meningitis, deceiving, however, only those who have not a thorough acquaintance with the whole course of the disease. Where a suspicion of meningitis exists, we cannot insist enough on the absolute necessity of a thoroughly trustworthy previous history.

Cases of meningitis have been observed up to the remarkably great age of sixty-three years. The lower limit of age disappears on account of the want of exactitude with which simple meningitis infantum and tubercular meningitis have been separated.

3. The diseases which might give rise to confusion are the following: cerebro-spinal meningitis, epidemic and sporadic. To distinguish it from this disease, causes the least difficulties; the severe initial chill, the pains in the nape of the neck and in the back, the appearances on the skin, the herpes labialis, the strange protean forms of the delirium, the repeated occurrence of the disease, indicate cerebro-spinal meningitis in such a way that the great majority of cases will be at once recognized. *Real*

improvement does not occur in tubercular meningitis; a long dragging of the disease, with remission and subsequent exacerbation of all the symptoms, which again disappear, a final recovery, exclude tubercular meningitis.

The slowly progressing forms of basal meningitis, as they will be treated of in the following section, may give rise to temporary confusion; only a longer observation of the course can then decide.

Simple *purulent meningitis*, which supervenes in phthisical affections of the lungs, can hardly ever be surely distinguished from the tubercular. In pneumonia and pericarditis the diagnosis, "simple terminal meningitis," may well prove correct; but we have seen this diagnosis made under the most favorable circumstances, and the disease turned out to be a tubercular meningitis of rapid course, having its origin in old unrecognizable foci of the apices. There is here, in fact, a limit, beyond which the diagnosis cannot go, since an accurate knowledge of all the smaller cheesy foci is impossible.

Several times the differential diagnosis could not be made in the beginning between congestive hyperæmia of the brain and meningeal tuberculosis; the symptoms may entirely coincide. There remains no other choice than accurate further observation, since in many cases it is impossible to form any conclusion as to the existence of cheesy foci.

Beginning tubercular meningitis is from time to time brought to the hospitals under the name of mania, melancholy, hysteria, delirium tremens. Such mistakes cannot, of course, last long; the conditions of fever alone would soon arouse the attention; moreover, the psychical lesions in meningitis do not preserve constantly or for any long-continued time the same character; they soon change to conditions of sopor, or there appears a facial paralysis, or a paralysis of the pupils—in one word, the eminently progressive character of the disease shortly furnishes a diagnosis. Delirium tremens alone may cause difficulty for a certain length of time, but only in the case of old decrepit subjects, in which cases death may occur through complication with concealed pneumonia or with some other inflammatory lesion, and with development of various brain symptoms. Especially in such cases as these an accurate examination of the previous history and of the etiology will prevent error.

Individual cases of old affection of the brain, arising on a basis of tuberculosis, are very difficult to diagnosticate—cases which may then be brought to a close by a tubercular meningitis (see the case already described). The physician who has the good fortune to observe many cases of this nature, will come to have a certain skill in the choice of probabilities ; but it will be hardly possible, even for him, to separate the individual series of symptoms in an accurate scientific manner. There will be called into question one after the other the possibilities of tumor (tubercle, cheesy focus), gumma, necrotic softening, inflammation in the form of simple encephalitis, or of abscess of the brain. We must call special attention to the final stage of abscess of the brain, which may have such a resemblance to tubercular meningitis that confusion is not to be avoided. The lists of patients at the Zürich Clinic show three such cases (Griesinger, Biermer, my own observation).

The sudden appearance of lesions of the speech must render a confusion possible with all those affections which have their point of origin near the cortex of the island of Reil, and which occasion aphasia. It can then assist us in making the diagnosis, when all the circumstances force to the hypothesis of a development of miliary tubercles in the body. We must, moreover, be sure of the healthy condition of the heart and of the vessels ; otherwise a large embolus of the brain, taking its origin in the heart or the vessels, may be found in that place in which we had thought to discover a partial meningitis with miliary tubercles.

Mistaking for uræmia has taken place in those cases in which a tuberculosis of the urinary organs has been confounded with a parenchymatous affection of the kidneys. The grounds for distinguishing affections of the kidneys, are to be found in careful preliminary observation and in examination of the urine. An abnormal course of uræmia may indeed have a certain resemblance to meningitis, but in the majority of cases the convulsions and the whole course of the disease give sure distinctive signs.

Real difficulties further arise in the case of caries of the petrous portion of the temporal bone. It is well known that thrombus of the sinus, as well as simple purulent meningitis, frequently gives rise to appearances which, considering the known variety of forms taken by tubercular meningitis, may very readily coincide with these latter. If there is suspicion of general tuberculosis, or if this can be positively shown as a localized affection, it must then be considered whether the otitis may not perhaps be entirely innocent of the existing brain symptoms, and whether we have not to do with general miliary tuberculosis of the pia mater. Here, too, the circumstances may be such as to prevent us from distinguishing.

It is very difficult to distinguish between tubercular meningitis and leptomeningitis infantum, spoken of above. A series of cases are indeed not to be distinguished from each other.

In some cases the etiology may be of decided assistance in distinguishing. All the circumstances, which have been claimed above as causal grounds of leptomeningitis infantum, will play no part in tubercular meningitis. On the other hand, in this latter, constitutional anomalies are worthy of the most serious consideration. It is clear that these diversities of origin cannot decide the matter in all doubtful cases.

Since tubercular meningitis is observed as early as at the beginning of the second year, the age cannot decide the question, for it is here only a question of children in the earliest youth.

In the development of the symptoms both affections vary between about equally wide limits ; the succession, the intensity of the symptoms of disease seem to us to furnish really no points on which to make a differential diagnosis ; it is only that in simple meningitis of children the convulsions seem to be more frequent and more intense ; still no positive decision will be risked.

In simple meningitis a prodromal period is wanting. This prodromal period is therefore of great importance if it can be really proved, especially if it occurs between an acute affection (pneumonia, typhoid, etc.) and a new disease with corresponding brain symptoms. It is, however, absent in many cases, or its place is taken by an indefinite and not easily explicable simple decrease of nutrition.

The result may instruct us as to the nature of the affection ;

we see cases of simple meningitis recover, leaving more or less traces; not so tubercular meningitis.

The resemblance of tubercular meningitis to typhoid has been shortly spoken of above. Special care must be taken, in doubtful cases, not to insist on putting forward the character of the delirium as characteristic of the one or the other affection, since no positively characteristic sign is to be found in this, according to the observations of those who have seen many irregular cases of both categories. It is moreover to be observed that we cannot consider as a characteristic sign of inflammation of the pia mater the rigidity of the muscles of the nape of the neck, of the back, or of the whole body. This occurs also in cases of typhoid with severe disorder of the brain. The same may be said of spasmodic shiverings, and indeed of general convulsions. Constipation occurs in both diseases; much oftener, it is true, in meningitis, but we have seen precisely the worst cases of typhoid run through their course with obstinate constipation. Retraction of the abdominal muscles, if well developed, is a good sign of meningitis; but, on the other hand, cases of meningitis do occur, accompanied by diarrhœa, and with no retraction of the abdomen.

The curve is not conclusive; the course of temperature in severe typhoid often shows great departures from the normal. Intestinal hemorrhages indicate typhoid; but bloody stools—where there were tuberculous ulcers of the intestines and no affection of the lungs—once led us to suppose the existence of typhoid, when meningitis really existed. Well-developed typhoid roseola is perhaps the best indication of typhoid; slightly developed exanthematous roseola has also been observed in tuberculosis of the pia mater. According to what has been said before, even the condition of the eye may lead us astray, unless there is marked neuro-retinitis. It is needless to say that these errors of diagnosis occur in cases which vary from the normal type.

Finally, there are cases of septicæmia which may lead to confusion with tubercular meningitis. A certain class of these has aroused great interest of late; these are those acutely occurring processes of inflammation in and upon the bones of the extremities which may lead to a rapid absorption of poisonous matter, to numerous small inflammatory foci in the lungs, to fatty embolism of the lungs, to endocarditis of unusual nature, and from this again to multiple arterial embolism (skin, brain, etc.).

Some cases have been recognized to be of mycotic origin (Eberth). The disease is of a very severe type; sensorium early and seriously involved, delirious attacks, groaning, screams, quickly ensuing sopor and coma; no symptoms whatever of foci in the brain, or only the faintest; very high fever; rapid pulse; in some cases affections of the lungs and heart; the primary point of attack of the disease generally assignable to a painful swelling of an extremity (humerus, femur); finally, but not in all cases, small extravasations under the skin, like petechiæ, capillary embolisms in the skin and mucous membrane, mostly in common with such in other organs. The disease has given rise to very frequent confusion with severe typhoid (enlarged spleen in both). In one case the diagnosis was septicæmia, and the autopsy showed acute miliary tuberculosis (without that of the pia mater); in a second case the diagnosis was miliary tuberculosis, and the autopsy showed septicæmia. Mistaken diagnoses of these diseases will continue to occur, especially in hospital practice, owing to the limited time for observation, since this form of septicæmia is not frequent, therefore not thoroughly studied, and the symptoms such that, without better means of observation than now exist, it is, from the nature of the case, simply impossible to distinguish it.

Prognosis.

The opinions of all physicians agree in this, that tubercular meningitis, at whatever time of life, is one of the most dangerous affections; the opinions vary as to the answer to the question whether the disease is always and under all circumstances fatal. Before anything else, it should be here mentioned that simple observations at the bedside, which are not subsequently subjected to the test of the autopsy, deserve no sort of attention; whoever compares the symptoms of the disease under discussion with the symptoms of other forms of meningitis very soon discovers how illusory is the attempt intra vitam to distinguish them in a manner absolutely correct in all cases. Now as leptomeningitis infantum is not necessarily fatal, and as other forms of meningitis may, under certain circumstances, be cured—generally, it is true, leaving traces behind—we may reasonably infer that the cases reported as cured were really *not* cases of tubercular meningitis. We do not by this mean to throw any doubt upon the accuracy of the observations of Hahn, Rilliet, and others, which show the principal symptoms of the disease in progressive development, but it is allowable to doubt the correctness of *diagnosis.*

Every day teaches that the most practised diagnosticians are not always able to avoid mistakes in this field.

It would be quite otherwise, when one had an opportunity at times to see the positive remains of miliary tubercles in the cadaver. But pathological anatomy furnishes no information of the correctness of which it would not be possible to doubt. Individual observations, which have been held up as instances of cured miliary tuberculosis of the pia mater, are not supported by the necessary patho-anatomical, that is to say, histological proofs. With all respect for Hasse's care and thoroughness, we are at variance with his belief in the possibility of a cure of miliary tuberculosis of the pia mater, and we have never found in the pia mater anything resembling a harmless encapsulated deposit, or indeed traces of absorbed miliary tubercles. We do not, therefore, consider an ending in recovery as proved, either by the observations on the living or by post-mortem examinations. All the cases of our own observation—by no means limited—in which the diagnosis was well grounded, resulted in death. One case, of which we spoke at the time as cured tubercular meningitis, proved to have been (the child subsequently dying of pneumonia with cheesy degeneration) simple serous effusion; neither fresh miliary tubercles nor traces of absorbed tubercles were to be found either in the pia mater or elsewhere.

It remains undecided whether a very limited number of miliary tubercles might remain without doing harm, and might be subsequently removed by some retrograde process; observations in point are wanting; all the cases of partial miliary tuberculosis which we have seen led to death, either suddenly or by occasioning in this neighborhood dangerous processes of inflammation; in the latter case, it is true, death occurred after a somewhat protracted illness.

Treatment.

Since we know of no cures of tubercular meningitis, the important point of treatment for us is not what is to be done after the disease has declared itself, but what can be done to prevent the same. At the same time we have taken the ground that there are types of disease which entirely coincide with the type of tubercular meningitis, but which belong to another form of meningitis of which the prognosis is better. That is, we are by no means authorized to limit ourselves in all cases to making a diagnosis and prognosis; but it is our duty in each case to use those measures which, according to general consent, are capable of alleviating certain symptoms of disease.

The prophylactic treatment would be one of the most satisfactory parts of the physician's practice, and productive of the best results, were it not for outside circumstances, which come to be placed as hindrances at every step in the way of its rational accomplishment; a general imitation were to be wished for of the practice of that city of Lombardy which sends to the sea-coast, for a time during the fine season, the scrofulous children of its poor. But aside from this there is a field in which the activity of the physician can work blessings. Where scrofula and tuberculosis have taken up their abode in families, we must go to work against them with all the means in our power; mothers who have been scrofulous should not give the breast to their children, nor should mothers whose previous diseases, or whose conditions of nutrition lead us to fear the development of tuberculosis; scrofulous children should be sent away from the city or place of their birth; anomalies of nutrition should receive the treatment which is known to be really useful in such cases (cod-liver oil, iron, iodide of iron, salt baths, sea-shore and mountain residence). It is remarkable that change of climate has often the best results where we cannot say that the new conditions of hygiene are really better than the old. A proper diet should be prescribed, and, if possible, all improper articles of food should be avoided. Too early mental activity should be prevented; all chances of taking whooping-cough, measles, diphtheria (schools for young children), should be shunned, and these primary affections thus kept off, which so often change scrofula to tuberculosis. Bronchial catarrh should be treated with the greatest care; affections of the bowels as well; and in all these disorders, more than the necessary care should be exercised. We should endeavor, by all the means in our power, to secure the establishment of good hospitals for children, and facilitate their use in every way.

It is very strongly to be advised that all swollen lymphatic glands which can be reached from without should be removed by operation; the young organism is thus spared a long suppuration, its most dangerous enemy; we cannot, unfortunately, approach the internal glands which may be in a state of cheesy degeneration.

The treatment of pronounced meningeal irritation and of the succeeding symptoms of compression is unfortunately, even at the present day, only the old treatment of brain symptoms to be modified according to the special circumstances. The following measures must therefore here be mentioned once more:

1. *Taking of blood from the head.*—No one ever speaks of (general) bleeding at the present day; the taking of blood must in all cases be simply local. Leeches on the temples and on the mastoid process, cuppings on the nape of the neck, often have a good effect, which, though undeniable, is unfortunately only temporary. The sensorium is clearer; the pains are less violent. The good effect lasts, however, but a short time; the cloudiness of mind soon returns again; and, if we have to do with miliary tuberculosis, a repetition of the taking of blood has, as a rule, no further effect. The action of (general) bleeding in compression of the brain occasioned by hydrocephalus is entirely illusory, and only hurries the patient more quickly to his end.

2. *Derivatives.*—The application of these to distant parts of the body has long since lost credit. The application of mustard poultices and blisters to the calves of the legs, mustard plasters, rubbings with mustard spirit, etc., are now only ordered to

satisfy in practice outside demands. Care must be taken not to go too far with these things, which, according to experience, do not even afford temporary relief.

Derivatives locally applied on the head have not helped us at all. Blisters near the head or upon it should be entirely discarded. Painting the shaved head with iodine, although energetically applied, was quite without result. Applications of croton oil and of antimonial ointment on the head have come to be obsolete.

Intestinal derivatives are of much more value, and are not to be neglected. Copious discharges should be obtained. The much-abused calomel will do its work as well as any other purgative, if the necessary precautions are not neglected. In one case where the physician continued to give calomel, although not obtaining the wished-for effect, we saw a very sudden appearance of stomatitis and double parotitis, which actually hastened the end. We therefore prefer to use the vegetable purgatives (castor-oil, senna), in connection with salts. The administration must be continued till its full effect is obtained, since it is only in this way that any result may be had from this treatment. In the beginning of the disease copious stools are observed to give relief. Where great effusion continues, a direct lessening of the effusion has been expected to result from this method; and, with this object, diuretics have been used as well as purgatives. This opinion may be described, at the best, as very well intentioned.

3. *Cold.*—The ice-bladder is still the most convenient mode of using cold. Other methods of application (irrigation, ether spray) may be tried; we have seen no advantage from their use. The application of cold has been abandoned by more than one. Hasse advocates warm applications, following Romberg's example in meningitis of the convexity. We should not wish to give up the ice-bladder.

4. *Quicksilver and preparations of iodine have been employed as real specifics.* Calomel, corrosive sublimate, blue ointment, iodide of potassium, iodide of mercury, iodine ointment, iodide of potassium with calomel and digitalis, have been used a hundred times, extolled, and again abandoned with disappoint-

ment, as deceptive. They are certainly entirely useless in tubercular meningitis. We do not maintain that the mercurial treatment is absolutely useless in simple meningitis; our experience does not enable us to decide this question. In any case, care must be taken to avoid salivation.

We may be allowed to pass over in silence the lists of specifics which have made their appearance for a moment and have immediately disappeared again.

5. *Cold douches and compresses.*—Cold douches in a lukewarm bath are the most vigorous means of temporarily rescuing patients from coma, and have done us good service in some cases with children, where there was no question of tubercular meningitis; in such cases they were repeated hourly. We are really disposed to ascribe to these a great influence on inflammatory processes in the pia mater. They are to be employed in states of coma, and are especially useful in practice among children in eclamptic convulsions following coma. If we have to do with tuberculosis of the pia mater, these do not, it is true, give any permanent good result; in all other forms of meningitis, especially in children, we are their enthusiastic votaries. It must be added that they display at the same time an antipyretic action. The same may also be said of cold compresses, which, beyond this, show no action worthy of being praised. To bring the patient out of a state of coma, energetic douches are certainly far to be preferred to any internal medication.

6. *Stimulants.*—A sort of moral sense of duty compels us to employ these towards the close of life. Musk, camphor, ammoniacal and alcoholic preparations, phosphorus, arnica, valerian, asafœtida, preparations of zinc, are employed without the slightest result; as momentary stimulants, preparations containing alcohol, and musk, are entitled to mention.

7. *Narcotics.*—Hasse and others advocate these with the greatest reason. In fact, small doses of opium, morphine, and chloral are indispensable in cases of great jactitation, delirious uneasiness, screaming, objectless movement, etc.; these will, however, never prove of any other value than as palliatives. We have proved the preparations of bromine to be entirely without effect.

8. *Antipyretics.*—There is seldom a call for the employment of these in tubercular meningitis. They are, however, to be used energetically in case the diagnosis should be doubtful as between meningitis and typhoid, or septicæmia. Here the treatment to be followed would be the use of cold baths, coupled with large doses of quinine, salicylic acid, salicylate of soda.

III.—*Simple Meningitis of the Base.*

Processes of meningitis of the base, without a foundation of tuberculosis, are more unusual forms of disease. These consist in reality of slowly or quickly progressing inflammatory lesions of the pia mater of the base of the brain, from the lower surface of the frontal lobe down to the pyramids, lesions which are limited in some cases to the formation of dense, hard sheets of connective tissue, which are capable of injuring the neighboring delicate organs in their texture and their functions, but in other cases give rise to more acute exudation, which appears as a moderate, fibro-purulent infiltration of the tissue of the pia mater and that of the plexus. In rarer cases the whole process is so acute that the disease resembles, in many respects, tubercular meningitis, so much so that mistakes occur, which, in some cases, are not to be avoided.

In regard to the pathological anatomy, we would call attention to the following points:—

The changes at the base of the brain are very different, according to the acuteness of the process. The most acute case which we had the opportunity of examining (course: seventeen days) showed marked fibro-purulent infiltration of the pia mater of the base, from the chiasma quite to the posterior limit of the pons; this (infiltration) entered the fissure of Sylvius and extended along the internal carotid arteries, on the upper side of the cerebral branch, running on the upper surface of the caudex cerebri (Hirnstamm), but did not reach the convexity of the brain. It was easily to be followed along the whole scissura transversa of the cerebrum from one gyrus uncinatus to the other, and involving the plexus. The oculo-motor and abducens of the left side were surrounded by thick purulent sheaths; the other nerves were more or less covered with purulent matter. There was hydrocephalus in a very high degree; the liquid was remarkably rich in cells; strong distention of all the ventricles, of the aqueduct of Sylvius, of the fourth ventricle; hydrocephalic softening, noticeably slight; no trace of miliary tubercles either at the base or in the pia mater of the convexity, from which the blood had been pressed out so as to render it anæmic. Opposed to such cases of acute change are others, which evidently occur much more slowly: the inflammatory infiltration has led to various degrees of thickening of the pia mater; the grayish white organized plates of connective tissue, between which a moderate degree of suppuration is recognizable with the microscope, are to be considered as old products; it is evident that extravasation of the white elements of the blood and the accompanying organization into cells is a relatively slow process. The plexuses are found in a very characteristic condition of induration, increase of size, and inflammatory infiltration which, during an acute period, may very well lead even to a new coating with pus.

The last-named cases are those with which we shall have principally to occupy ourselves. There appears, finally, in a third category of cases, processes entirely chronic, which lead to the formation of cicatricial, much indurated, connective tissue, whose extent is often extremely limited.

These three categories of cases are so related to hydrocephalus that, in the first two, this is present, but is wanting in the entirely chronic, connective-tissue changes. It seems to follow from this that the cases of the last category are to be considered from an entirely different point of view, and it is indeed very probable that they are, from first to last, inflammations *whose cause goes over from the bones to the pia mater;* they should not, therefore, be treated of at this place.

We easily recognize, too, in this hydrocephalus the principal cause of death; so that in the cases of the first category an acute or subacute process seems easily conceivable, in the second category a chronic or nearly chronic course.

Where hydrocephalus is present, it is generally very severe. All the prolongations of the lateral ventricles are distended very greatly, the aqueduct of Sylvius and the fourth ventricle as well; the inflammatory infiltration of the plexuses has already been mentioned. Quite differently from what occurs in the forms of acute hydrocephalus previously described, in these forms the ependyma does show alterations; for the most part, there are thickenings of the ependyma, granulations of the ependyma, frequently not on all parts of it; this is accompanied not very unfrequently by a hydrocephalic liquid, rich in cells, which cells may even have been deposited on the walls of the ventricles, like a grayish-yellow covering. A chronic ependymitis has, therefore, also been spoken of here. The examination of the ependyma allows us to recognize the well-known connective-tissue thickenings, but never anything like real inflammatory alterations.

Parallel with the alterations of the ependyma, is found an absence of hydrocephalic softening, or, at least, this is only very slightly developed. Severe hydrocephalus leads, however, as regards the brain, to results analogous to those which occur in tubercular meningitis; it occasions pressure on the hemisphere; the cortex is pressed as closely as possible against the inner surface of the dura mater. The consequences are such as have been above described, viz., narrowing of a great number of vessels, and hindrance to the normal entrance of the flow of blood. We find, therefore, here, too, the substance of the brain dry and relatively anæmic, the gyri flattened, the sulci obliterated, the pia mater without fluid in the subpial space and as if pressed out, dry and without moisture.

We are not authorized to form a judgment as to the more delicate alterations of the cortex. In the single case which we hastily examined, the cortex of the convexity was entirely free from inflammatory alterations; no exudation of white blood-globules, etc., to be discovered. This may vary very greatly according to the extent and acuteness of the process. Some reports of autopsies tell, in fact, of slight extravasations of blood, reddening and discoloration of the cortex, especially in the neighborhood of the alterations at the base.

Those alterations of the pia mater at the base, which are decidedly of a chronic nature, where there is absence of diffuse, widespread changes in the cortical substance, do not imply any hydrocephalus ; but we have already suggested that these cases should, perhaps, be wholly relegated to another place. They are noticeable in this, that here the nerves of the brain have been frequently found injured in a very marked manner. Strangulation of the oculo-motor, of the abducens, of the facial, even paralysis of *several* nerves of the face together, may occur.

It is clear that in such affections of these nerves, without the general action of hydrocephalus, without progress of inflammatory changes on the convexity, and without real alterations of the cortex, there must result very remarkable types of disease. Without doubt, specific processes of the bones play the principal part in these cases.

The behavior of the other organs shows not the slightest regularity, quite differing in this from what is observed in tubercular meningitis. In the lungs bronchitis and its sequences have been cited, broncho-pneumonia and atelectasis, lobar pneumonia ; then, again, an entirely healthy condition, or a condition showing positively no connection with the disease in question. There is no question of any coincidence with tuberculous alterations, and in the reports of autopsies to which we have had access there has been, as it happens, not one where there was any proof of cheesy degeneration of a lymphatic gland anywhere in the body. Nothing has ever been found in the heart which would throw the least light on the etiology of the disease. In like manner, stomach, intestines, and all the abdominal organs have always been proved entirely healthy. Liver and kidneys have showed nothing beyond anomalies in the distribution of the blood. The spleen has never, at the autopsy, been found noticeably enlarged. No affections of the bones have ever been observed, *except the above-mentioned cases, with an entirely chronic course.* The only processes with suppuration which are mentioned are : in one case, a gonorrhœa ; in a second, a slight suppuration in the left epididymis ; in a third, a suppurating slough on the thigh.

From time to time there may be found, it is true, a more specific cause (fissures at the base, tumors of the base, abscesses of the brain, etc.) for inflammatory processes at the base of the brain. We shall speak further on of this form of inflammation, since it has no connection whatever with spontaneously occurring meningitis of the base, the causes of which are clearly quite separate, though entirely unknown.

It is noticeable, and is perhaps more than the result of chance, that nearly all the patients are in the force of youth; sixteen and thirty years are the limits of age. The cases cited have been, as a rule, not of cachectic, but of strong persons, in good conditions of nutrition. Even during life it was generally established that tuberculosis had nothing whatever to do with causing disease—that is to say, no hereditary condition bearing upon the disease could be discovered.

With individual patients, great exertions had preceded the commencement of disease; no connecting link was, however, perceptible. Almost all the cases were of workmen (masons, butchers, locksmiths, farm laborers, waiters, etc.). The nature of the employment, however, gives no explanation of the different kinds of injury.

Duration.—Very remarkable and enigmatical is the average duration of the disease (seventeen to sixty-four days). The case of shortest duration known to us (our own observation) resulted in death on the seventeenth day; that of the longest course (Biermer, cited in Seitz, hydrocephalus) lasted sixty-four days. Between these there are cases of twenty-two, twenty-eight, forty-three, fifty-eight, sixty days (Tüngel). The course of the disease is marked by great variability in the intensity of the symptoms, doubtful improvement, with quickly following relapses. Some cases, in which the diagnosis seemed thoroughly to be trusted, came to be cured.

23. Butcher, twenty-five years old, strong, healthy; had worked for a week together in an ice cellar; had felt somewhat dull and unwell for some days.

October 7th.—Severe headache; vomiting.

October 8th.—Admitted to hospital, with very severe headache; pains in the limbs; want of appetite; tongue furred; stools sluggish. Temperature 37.2° (99° F.). Pulse 72. Strong sweats; miliaria rubra; chest normal; abdomen also; pupils equal; movements unhindered; dullness of hearing; nausea and eructation; restless night.

October 9th.—Headache very violent; groaning, screaming; increase of deafness; vomiting; pains in the neck and in the body. Temperature 37.5° (99.5° F.). Pulse 72. Temperature 38° (100.4° F.). Pulse 88, irregular; quiet night.

October 10th.—Loud complaints of headache; thin stools. Temperature 37.9° (100.2° F.). Pulse 72. Temperature 38.6° (101.5° F.). Pulse 84. No vomiting; no change in the pupils; movements unhindered.

October 11th.—Renewed headache, so violent as to render narcotics necessary; no difference between the two pupils; paresis of right facial. Temperature 37.9° (100.2° F.). Pulse 72.

October 12th.—Temperature 37.2° (99° F.). Pulse 84. Paresis of right facial; power to close the eyes unimpaired; pupils equal; pressure of the hand the same on both sides; tongue somewhat inclining to the right; retention of urine, rendering the use of the catheter necessary; headache very severe; dullness of hearing; no vomiting. Temperature 38.8° (101.8° F.). Pulse 64.

In the evening.—The facial paresis becomes less marked.

October 13th.—Temperature 37.5° (99.5° F.). Pulse 78. Temperature 39.5° (103.1° F.). Pulse 84. Violent headache; movements unhindered; pupils equal; facial paresis not observable any more; marked sinking in of the abdomen; twitching of right side of the face in sleep.

October 14th.—Temperature 38° (100.4° F.). Pulse 80. Temperature 39.4° (102.9° F.). Pulse 82. On the right testicle is marked tenderness to pressure, without actual swelling; condition otherwise the same; violent headache, on account of which the administration of opium is continued.

October 15th.—Temperature 37.7.° (99.9° F.). Pulse 84. Temperature 39.5°–40° (103.1°–104° F.). Pulse 100. Up to noon no headache; this returns in the afternoon; less dullness of hearing; stool normal.

October 16th.—Temperature 36.7° (98° F.). Pulse 80. Temperature 39.2° (102.6° F.). Violent headache in the afternoon, against which quinine is employed.

October 17th.—Temperature 37.3° (99.1° F.). Pulse 64. Temperature 38.3° (100.9° F.). Same condition.

October 18th.—Temperature 37.6° (99.7° F.). Pulse 84. Temperature 39° (102.2° F.). Pulse 94. Violent headache.

October 19th.—Temperature 37.6° (99.7° F.). Pulse 64. Temperature 39.2 (102.6° F.). Pulse 90. Very intense headache.

October 20th.—Temperature 37.4° (99.3° F.). Pulse 70. Temperature 39° (101.2° F.). Same condition.

October 21st.—Temperature 38° (100.4° F.). Pulse 64. So far from there being as before a dropping of the left corner of the mouth, there is now twitching of the muscles of the right corner of the mouth; frequent grinding of the teeth; toward evening the headache is severe beyond measure. Temperature 39.4° (102.9° F.).

October 22d.—Headache. Temperature 38° (100.4° F.). Pulse 76. Muttering, lying half awake; grinding of the teeth. Temperature 39° (102.2° F.).

October 23d.—Paresis of the left facial; nothing to be observed in pupils; sinking in of the abdomen; hearing better; urine passed; stool following clyster. Temperature 37.9° (100.2° F.). Pulse 96. Temperature 37.3°–37.7° (99.2°–99.9° F.). Delirious attacks in the night; twitchings of the extremities.

October 24th.—Temperature 37.5° (99.5° F.). Pulse 60. Temperature 37.5° (99.5° F.). In the forenoon sleep; then complete mental disarray; screaming; talks much with himself; sings; gnashes his teeth; involuntary passages; no appearance of violent pain.

October 25th.—Temperature 37° (98.6° F.). Pulse 66. The mind is wandering; he does not recognize those about him; he is delirious, noisy, and resists; the pupils equal; left abducens paralyzed. Temperature 38° (100.4° F.). Pulse 66.

October 26th.—Temperature 37.5° (99.5° F.). Pulse 84. Condition the same; paralysis of left facial and left abducens; twitching of the right half of the face. Temperature 37.5° (99.5° F.). Is conscious for two hours; then the former delirious attacks return.

October 27th.—Temperature 38° (100.4° F.). Pulse 88. Twitchings on various parts of the body, succeeding each other without regularity; right pupil dilated; involuntary passages; delirious attacks; wants to tear his shirt, etc. Temperature 37.7° (99.9° F.).

October 28th.—Tonic contraction of the muscles of the face, of right thumb, of right great toe; grinding of the teeth; the former paralyses; consciousness entirely gone. Temperature 38° (100.4° F.). Pulse 108.

October 29th.—Temperature 38° (100.4° F.). Pulse 132. Tracheal râles; absence of consciousness.

October 30th.—Temperature 38.5° (101.3° F.). Death.

Autopsy.—Body emaciated; dura mater stretched; arachnoid of the convexity cloudy; convolutions of the brain less distinct; pia mater of the base, especially about the chiasma, infiltrated partly with serum, partly with yellowish clotty exudation; ventricles dilated, filled with opaque serum; in the posterior cornua soft yellowish clots of purulent appearance; ependyma somewhat thickened, only slightly softened, even at the fornix; granulations in the fourth ventricle, and on some portions of the anterior cornu; the brain moderately well supplied with blood; spleen small; in the left epididymis suppuration of slight extent; no other appearances of any importance. (Tüngel.)

Course of the disease.—The disease was at the beginning in all cases quite acute; it is seldom that mention is made of a short period of slight illness lasting only a few days. Usually the disease begins with general fever symptoms: languor, depression; then, in most cases, there very soon follows a chill, or at least chilliness, subsequently heat, sweating, thirst, entire loss of appetite, total unfitness for work.

The symptom which now comes to the foreground, and predominates during the whole course of the disease, is a very intense headache, affecting the whole head, at times more especially the occiput. In some cases this persisted during the whole fifty or sixty days of sickness, and increased to such an exorbitant extent as to make the administration of narcotics in large doses an imperative duty; in other cases it showed marked remissions. The irregularity in the march of all other symptoms of disease is remarkably characteristic during the whole course. Periods of entire consciousness alternate with mild or pretty acute delirious attacks; immediately before death the patient often sinks into complete coma. The same may be said of the motor symptoms, the paralyses, spasms, contractures. One paresis may disappear, or may give way to another; contractures and spasms appear for some hours, and then disappear again, and it is, above all, surprising that the motor symptoms only make their appearance at a very advanced period. But in some cases these have been entirely wanting, and the type of the disease is then at times exceedingly monotonous. The patient becomes reduced to a skeleton. Death follows during coma.

Individual symptoms.—The disease is characterized by pyrexia (see the case cited above); particularly the initial febrile movements are often very violent, the temperature rises in an irregular manner to 40° (104° F.) and above, returning in the morning or forenoon with a sudden leap to the normal point; from this suspicion may arise of irregular intermittent. But it is not all cases which show these high temperatures. The fever generally continues during the whole course of the disease; in the later stages, however, there are in some cases hours and days of entire freedom from fever. A third series of cases show, during the whole course, many periods of remarkably low, frequently even subnormal temperatures. There can, therefore, under such circumstances, be no question of any recognizable law governing the movements of temperature, and this has, up to this time, seriously increased the difficulties of diagnosis. The initial temperatures have very often caused the diagnosis to incline to a suspicion of typhoid disorder.

The pulse behaves very differently in different cases; in general it follows the temperature; but in most cases other influences may easily be observed to have their effect on the quality of the pulse. We very often see a marked sinking during the progress of the disease, an intimation of the pulsus cephalicus. A sinking of the pulse at the close of life to 48, mentioned in one of Tüngel's cases, must be considered as an exception, since, in the majority of cases, the pulse increases toward the end of life to an extraordinary degree—as in all processes of meningitis, becomes irregular, and finally intermittent.

The psychical symptoms also show remarkable differences when compared with those of other forms of meningitis. In a few cases (Biermer) the sensorium was entirely normal during the whole course, there was never any delirium, memory and judgment good until toward the end, when, with the general sinking of the forces, a general weakness and loss of energy overmastered the intellectual powers as well. Other cases, however, point to a lessening of mental energy even in the beginning—patients are incapable of giving information as to what has gone before. Slight delirious attacks occur in dozing, and this at the very beginning of the disease; then there come states of sopor of short duration, from which, however, the patient may be aroused if loudly called to, and on awakening immediately answers correctly again. This may continue with varying changes during weeks, until finally, with rising pulse, coma often arrives almost suddenly. The manner of death is not, however, by any means always determined by this latter, and in a few of the cases the mind remained responsive quite to the end. We hear exceptionally of strong delirium, of restlessness, of confused, quarrelsome conditions, of morose, capricious, irascible dispositions; but this is only passing, and gives way to milder forms of delirium. Some patients may appear in a thoroughly normal condition of mind, but on going into particulars, and especially on continuing the conversation beyond the limits of an examination of the patient, we find that they show marked mental inferiority; (thus, in one case observed by us, a patient, who seemed for some time in a normal condition of mind, mistook the members of his own family, and was not able to solve an easy problem in arithmetic). Those violent delirious attacks, those stupid, melancholy conditions, which are seen in abnormal cases of tubercular meningitis, do not occur in this disease.

With the continuous excessive headache, giddiness is either present from the very beginning of the disease, or occurs at some period of its course; at times there seemed to be some connection between this and the vomiting, paroxysms of the former preceding the latter.

The motor symptoms are by no means so frequent and varied as we might conclude from the results of anatomical examinations. In the first place we would once more state that these symptoms are either entirely absent during the whole course of the disease, or occur late and in limited number. Total paralyses of the larger groups of muscles have not been observed. The pupils often behave in an almost entirely normal manner during the whole course of the disease; they are sometimes cited as rather wide open; their responsiveness is sometimes sluggish; this subsequently becomes better once more, and is only entirely lost at the very close of life.

We have never seen a mention of the dilatation *ad maximum* of one or both pupils; but we have observed imparity in the last days of life, and, immediately before death, even contraction with incomplete responsiveness.

Paralyses of the muscles of the eye have likewise been seldom mentioned; sometimes paralysis of the abducens. Simultaneous paralysis of all the extrabulbar muscular branches of the oculomotor nerve is very infrequent. In many descriptions particular stress has been laid upon the fact of the integrity of all the muscles of the eye up to the time of death.

In regard to the facial nerve, it should be observed that paralyses of this are mentioned as being generally unimportant —indeed, hardly noticeable; they may even disappear in the course of the disease, and subsequently a definite paresis may attack the other facial nerve. The same is true of the hypoglossus, which in very rare cases was found paralyzed only on one side. Paralyses of the extremities, to the extent of entire abolition of the functions, are never mentioned. On the other hand, mention is made of weakness of both legs and interference with the gait of a paretic nature. Responsiveness to irritation of the pharynx disappears towards the end of life, as is the case in similar affections. It may temporarily disappear and again return, if the condition of sopor improves.

Of spasmodic symptoms, there occur: rigidity of the nape of the neck, and thrusting the head back into the pillow, yet not in all cases; and it is often expressly mentioned that rigidity of the nape of the neck and of the muscles in general was entirely wanting, and this in cases which otherwise showed all the symptoms in their full development. Moreover, some patients, on account of the intense headache, hold the head as motionless as possible, do not turn it, and never nod, on account of the unbearable increase of pain. This may lead to belief in the existence of rigidity of the muscles of the nape of the neck. Some patients enjoy marked relief on keeping entirely quiet; for this reason, on rising or walking about, they keep the face turned upward. Contractures in other muscular regions are also of rare occurrence; in most cases they are entirely wanting. Rigidity has been cited of certain groups of muscles of the fore-arm, the upper arm, and leg.

Clonic twitchings of the muscles have been sometimes seen, but belong decidedly to the exceptional symptoms. One case of Tüngel showed general convulsions. Grinding the teeth is very frequent during sleep or when half awake, and in the later stages trismus, so that nothing more can be administered by the mouth. Finally, there occur spasms of the diaphragm (singultus).

Sensibility or *sensitiveness.*—We do not find any notice of anæsthesia or of dysæsthesia; on the other hand, there is general hyperæsthesia, so that the patient must not be handled, and he cries out loudly at every touch. The functions of the senses, so far as observations are possible, appear normal; in one case of Tüngel dullness of hearing is mentioned; in a second case difficulty of vision; this case showed marked distention of the veins of the retina. Beyond this, there are ringings in the ears, scintillations before the eyes, and perhaps some hallucinations of hearing.

Enlargement of the spleen was found in some cases; but this was always most insignificant, and the observers seem even to have been doubtful of its existence at times. At autopsies the spleen is found to be small.

Stomach and intestines.—Vomiting is one of the most common symptoms. It may begin at any period of the disease ; in some cases it appears very late ; in others it is an initial symptom.

It is remarkable that, in spite of the vomiting, patients still eat with good appetite at times, especially during remission of the pain and when the fever is less. The tongue is slightly furred, generally moist.

Constipation is a very constant symptom ; the treatment has generally to struggle against this stubborn difficulty. The retraction of the abdominal muscles is far from invariable ; with some patients, on the contrary, the abdomen is inflated ; with others there is, by turns, slight retraction and normal condition. Involuntary passages are usual during the coma.

Accurate examinations of the urine are wanting ; great departures from the normal condition certainly do not occur.

The condition of the skin shows little worthy of remark. In some cases copious sweats are cited, which in each case caused temporary relief of the headache. In no case was exanthematous roseola observed ; in very rare cases, herpes labialis.

Before death the patients reach a state of the most extreme weakness and emaciation. Bed-sores often follow ; and yet death long delays to free them from their most wretched existence.

The dilatoriness and variability of the symptoms correspond, without doubt, to similar diminutions and exacerbations of the inflammatory affection of the pia mater. The causes of these variations entirely escape our view, as has been already mentioned. It has likewise been stated to be in the highest degree improbable that the ependyma is actively involved in the hydrocephalus. It is in general true that the more an effusion into the brain cavities assumes a chronic form, so much the more frequent and distinct are the granulous changes of the ependyma. We shall not be justified in considering this as chronic ependymitis until indubitable appearances of inflammation of the ependyma have been positively shown. It is plain that with this non-committal treatment the matter will not be rendered any clearer, and it only remains to acknowledge this, that it is impossible to pronounce in a defensible manner either upon problematical preceding affections of the brain or upon chance constitutional anomalies.

The recession of the psychical alterations, the diminished violence of the delirious attacks in comparison with other forms of meningitis, may be assigned without doubt to the much less extensive lesions of the delicate tissues of the cortical substance; this is indeed assumed à priori, since only very partial examinations have been made. We ascribe the chronic headache to the ventricular effusion, which must exert a marked pressure on the exceedingly sensitive dura mater. For the time being, we are obliged to ascribe the paralyses to the affection of the nerves of the brain at the base; that many other circumstances are at the same time to be considered, is plain from the analogy of tubercular meningitis, but accurate anatomical grounds are wanting. The rarely occurring contractures are doubtless ascribable to irritation of the nerve-tracts which end in the spinal cord; we are quite as unable to explain in a strictly physiological manner the occurrence of convulsions here, as in the case of tubercular meningitis. It certainly is connected with the irritation of the ganglionic organs of the medulla oblongata. What has been previously mentioned holds good in regard to the result of examination of the retina.

Opposed to the type of disease which has just been described, are others which may be looked upon as exceptional:

a. Meningitis of the base of similar character to that described above, but with a more rapid subacute course. One such case, which came under our own observation, ended in death in seventeen days, showed in the nine days during which it was under observation the exact type of tubercular meningitis, was indeed diagnosticated as such, and the autopsy showed entire freedom from miliary tubercles and from any tuberculous alteration in the body. The symptoms of such cases correspond so exactly with tubercular meningitis, that no further remark seems necessary. The case mentioned showed stasis in the retina—a commencement of neuro-retinitis.

b. Meningitis of the base of a chronic nature, with formation of indurated patches of connective tissue, strangulation and lesion of the nerves of the base, but without hydrocephalus. If the above described cases border on the chronic, this is really true of the type now under consideration. These latter are rare; we can present no case of our own observation. Their principal symptoms are chronic headache, giddiness, paralysis of the oculo-motor with strabismus; ptosis and paralysis of the pupils; paralyses of the facial, hypoglossus, and abducens. Here there is very seldom paralysis of the extremities (of the left leg, M. Rosenthal). In one of Benedikt's cases the type of disease was exactly that of paralysis of the bulb (all the nerves of the base of the brain were surrounded by contracted connective tissue from the sella turcica to the occipital foramen). There occur here (Socin) disturbances of vision, which may disappear on the application of suitable treatment. These consist in general in a diminution of the accuracy of vision, and in evident diminution of the field of vision. The results of ophthalmoscopic examination point chiefly to varying degrees of "choked-disk." An extreme degree of "choked-disk" may coincide with marked diminution of the quantitative sensibility to light; lessened accuracy of vision and diminution of the field of vision may be present without actual lesions of the retina. The number of cases in which a sufficiently accurate post-mortem examination has been made is unfortunately very small up to the present time (Horner).

To distinguish such cases from tumor at the base of the brain gives rise to the greatest difficulties; especially will this be the case if the paralysis of an extremity occurs as well, so that the paralyses are perhaps crossed, and if, too, at times vomiting accompanies the headache and giddiness; in such cases even the examination of the retina gives no trustworthy distinguishing sign, since in chronic meningitis of this nature all those peculiarities of the papilla may be developed, which occur in case of tumor. An examination of the paralyzed muscle by electricity is quite as little able to furnish us a distinctive sign. This is in fact the region where the symptoms of paralysis of the bulb, of tumor at the base, and of chronic contracting meningitis of the base, resemble each other pretty closely. Tumor has indeed been mistaken for paralysis of the bulb (Bälz).

Diagnosis.

According to the description of usual cases, which has been given above, a diagnosis is possible. The following points are to be especially noticed:

1. The sudden beginning in the case of healthy individuals in good condition, without any tuberculous antecedents whatever; and here it is to be observed that this is only of value as corroborative evidence.

2. The long duration of the process, with symptoms continuing for weeks at a time, and subject only to trifling variations, and among which headache always has a prominent place.

3. The late occurrence of paralyses, their partial development, the rareness of spasmodic symptoms, the long-continued delirium of varying intensity, seldom violent, and with intervals of clearness.

4. In distinguishing from typhoid, we are to take into account the possibility of infection, further the absence of enlargement of the spleen, of roseola, of intestinal symptoms. In spite of this, serious difficulties may arise, since it is well known that typhoid does occur with intense headache, with stiffness of the nape of the neck, with absence of roseola, with no evident enlargement of the spleen, with very trifling intestinal or pulmonary symptoms. Long-continued observation will certainly permit us to distinguish, and is the only means of distinguishing, in such doubtful cases, which are, it is true, of rare occurrence.

5. To aid us in distinguishing the disease from epidemic cerebro-spinal meningitis, we are to consider the situation as regards an epidemic; beyond this there is the markedly less acute course, the long-continued clearness of the sensorium, the feeble attacks of delirium, the repeated alternations of states of coma with reawakening to a clear state of mind, the small number and slight intensity of the motor symptoms, the absence of any characteristic eruption on the skin. Herpes is a symptom to which undue weight must not be given.

6. In distinguishing from abscess of the brain, we must consider the absence of any grounds for such a diagnosis in the previous history, the non-existence of abnormal conditions of the body likely to lead to abscess of the brain (purulent and ichorous processes, affections of the bones, purulent affections of the lungs), the rareness of spasmodic motor symptoms, the absence of the characteristic march of the disease, without which we should not venture to diagnosticate abscess of the brain.

7. Tubercular meningitis is to be thought of so far as this, that exceptional rare cases of this disease do have nearly as protracted a course. Unless positive signs are present of a tuberculous cachexia, it is impossible to distinguish such cases.

Prognosis.

It is probable that this disease is not absolutely fatal; true it is that the majority of cases result in death. We are met here by the same difficulty as in deciding in regard to cases of cure of tubercular meningitis, namely this, that it is impossible to form a *positively correct* judgment from the observation of symptoms during life; but the danger of error is certainly less here than there. The possibility of regenerative changes cannot be denied on anatomical grounds, since the recognized lesions are not to be compared to those of tubercular meningitis either as regards their intensity or their compromising effect on the tissues. This is at once clear, if we consider the early injury to the lymphatic passages occasioned by the tubercles, as well as the various interferences with the circulation resulting from the deposit and growth of the same. We must further call attention to the absence of hydrocephalic softening, which points to a much less severe affection of the brain substance surrounding the ventricles; finally, to the condition of the cortex, which is not charged with miliary tubercles. From this theoretical point of view the possibility of cure cannot be disputed. In this connection an observation of Tüngel's is of special interest:

24. A mason, twenty-seven years of age; in his fourteenth year had a fall from a considerable height, but quite recovered from it after two days. In his eighteenth year had catarrh of the lungs; in his twenty-third year had pneumonia. Some time

previous to his admittance, gonorrhœa with swelling of the inguinal glands of the right side.

February 6th.—Severe headache; chilliness; loss of appetite; goes to bed at once; moderate diarrhœa; sleep disturbed; forthwith delirious attacks; flushed face; dry lips; tongue furred, moist; abdomen somewhat sunken in; spleen moderately enlarged; lungs healthy; dullness over the heart somewhat extended; pulse quiet and strong. Temperature 39.2° (102.6° F.). Urine without albumen.

Sleep restless; groaning; several times diarrhœa.

February 9th.—Temperature 39° (102.2° F.). Pulse 56. Temperature 39.2° (102.6° F.). Pulse 64. Same condition; in the night patient is delirious; much twitching of the muscles of the face and of the extremities; during the day he is conscious; pains in the head, the nape of the neck, and the back; a thin stool.

February 10th.—Temperature 39.2° (102.6° F.). Pulse 54. Temperature 39.5° (103.1° F.). Pulse 60. Night somewhat more quiet.

February 11th.—Temperature 38.5° (101.3° F.). Pulse 54. Temperature 38.5° (101.3° F.). Pulse 48. Condition somewhat better; quiet night.

February 12th.—Temperature 37.7° (99.9° F.). Pulse 56. Some appetite; severe pain in the occiput and nape of the neck; bending forward the head is very painful. Temperature 39° (102.2° F.). Local taking of blood at the back of the neck is without result; restless night; much twitching of the muscles of the face.

February 13th.—Temperature 38.2° (100.8° F.). Pulse 56. Temperature 38.7° (101.7° F.). Pulse 64. Pain in the occiput and nape of the neck; sleep; but persistence of the twitchings of the muscles of the face, and frequent starts of terror.

February 14th.—Temperature 38° (100.4° F.). Pulse 50. Temperature 38.5° (101.3° F.). Pulse 54. Headache continues; pupils somewhat dilated, but equal; consciousness clear; no paralysis; at night groaning and whining.

February 15th.—Temperature 38.2° (100.8° F.). Pulse 52. Temperature 38.5° (101.3° F.). Pulse 56. Headache the same.

February 16th.—Temperature 37.7° (99.9° F.). Pulse 58. Temperature 38.7° (101.7° F.). Pulse 60. Condition supportable.

February 17th.—Temperature 38° (100.4° F.). Pulse 56. Temperature 38.7° (101.7° F.). Pulse 68. Feels tolerably well.

February 19th.—Temperature 37.5° (99.5° F.). Pulse 60. Temperature 38.2° (100.8° F.). Pulse 76.

February 20th.—Temperature 37.5° (99.5° F.). Pulse 66. Temperature 38.2° (100.8° F.). Pulse 64. Renewed headache.

February 21st.—Headache, giddiness and nausea on standing up; sluggish stool; moderate appetite; otherwise no change. Temperature 37.2° (98.9° F.). Pulse 70. Temperature 38° (100.4° F.). Pulse 76.

From this time forward the condition was essentially the same; continual headache, sometimes very intense; consciousness entirely clear; no appearances of paralysis; moderate appetite; stool sluggish. Temperature in the morning normal, in the evening slightly higher; pulsations from 66–80. On the evening of the twenty-eighth steady increase of the headache, on account of which quinine was given, but without effect. At times during the night there are delirious attacks; twitchings of the muscles of the face.

March 1st.—Very severe headache, whining and groaning; sleep quiet, but on the following day a return of very violent headache; no fever. Now improvement; still there is every time headache and giddiness on sitting up; no actual emaciation; appetite good.

March 11th.—Bleeding at the nose giving temporary relief. Continual headache, until a seton is introduced, after which there is gradual diminution of headache and giddiness.

April 25th.—Leaves the hospital.

Treatment.

It is the physician's duty, not only to employ every means to alleviate the suffering of the patient, but to adopt every measure which offers even the slightest chance of making an impression on the inflammatory process in the skull, since the prospect is not so hopeless here as in the case of tubercular meningitis. In the first rank stand the local takings of blood, which are to be abundantly and repeatedly employed; the application of cold is agreeable to most patients, and they grasp eagerly for the ice-bladder; others on its use experience rather an exacerbation of the headache. The use of derivatives applied to the intestinal canal should likewise be sufficiently energetic, but rather less frequent. Blisters, issue and seton are without effect in the beginning of the disorder, but if there is a turn for the better, we may rather expect some good effect from the use of the seton. The employment of quinine has proved inefficient; the same is true of mercury in every form, and of the iodine cure likewise. All the neurotics glance off without effect, and indeed only those measures seem efficient which attempt to produce a lessening of pressure in the skull by a suction on the periphery. Still, experience does not justify too sanguine hopes.

The terrible headache demands a rather free administration of narcotics, opium, morphine, injections if necessary; chloral brings at least some sleep. The frequent vomiting likewise calls for the application of the appropriate measures, but these hardly prove useful even as palliatives. We must be cautious in judging the effect, especially of those measures employed against the inflammation, and must not deceive ourselves as to the amount of assistance which we may be able to render to the natural compensating action.

The rare cases of inflammation at the base, which are characterized by a lesion of the nerves there located, should be most accurately examined with a view to the existence of latent syphilis. In all cases, even where this latter is not to be proved by the previous history, a trial should be made of antisyphilitic treatment. The usual methods are here to be recommended: rubbings with mercurial ointment, iodide of potassium, both in connection with, or without the employment of Zittmann's draught; the most positive improvement has often been obtained by these means, even in those cases where it seemed necessary to exclude all idea of latent syphilis.

IV.—*Meningitis of the Convexity dependent on Neighboring Inflammatory Conditions.*

The forms of meningitis which come under this head, in general have their seat in that portion of the membranes which covers the convexity of the hemispheres, though their localization is not fixed and invariable, but depends upon the seat of the primary inflammation. Hence are their seat and extent very variable indeed. That form alone which springs from caries of the inner ear varies greatly, not only as regards the anatomical changes induced, but also in its intensity and localization; beyond this there is variation in its relation to other diseases—for instance, to thrombosis of the sinuses, which is a result of the same cause, and is associated with suppurative encephalitis, dependent on similar changes in the bony tissue. Thrombosis may even be dependent on encephalitis without having more than an indirect relation to caries of the petrous bone.

The very complexity of the etiology compels us first to glance at the inflammatory changes in the neighboring parts, which lead to purulent infection of the pia.

1. Diffuse meningitis of the convexity may ensue on acute inflammation of the bones of the skull, which inflammation, again, in the great majority of cases, depends on injuries and

wounds of all kinds, foreign bodies, and fractures, with or without injury of the soft parts. Here we have a confirmation of the observation made long ago, that acute inflammation of the bones, which extends further than the limits of the original lesion, arises when there is free access to atmospheric germs. Bones show a special tendency to become further involved when there are coagula on the exterior, which break down on the access of air ; and the same results follow coagula on the interior, provided that a fracture of the bone allows the air to enter. In such cases we have rapidly formed purulent deposits in the cancellar tissue of the diploë, between the skull and the pericranium, and finally between the skull and dura mater. It sometimes happens, when the primary injury of the dura was limited in extent, that its inner layers are protected from infection by the formation of healthy granulations ; if the injury, however, be of tolerable extent, the pus gets to the inner surface of the dura. But very frequently before this a delicate and more or less extensive adhesion has taken place between the dura and pia, over which, as over a bridge, the inflammation extends to the pia, and purulent meningitis of local origin, but very rapidly spreading in all directions, is the result. If, in addition to this—recognized or unrecognized—an abscess or a superficial purulent inflammation of the brain appear beneath, the injury was from the very beginning a complicated one—contusion of the brain. But the course is not always so simple, for the veins of the diploë often become filled with puriform and broken-down thrombi. These veins being in direct communication with the sinuses of the dura, the malignant thrombosis can easily extend into the longitudinal and transverse sinuses, and result in genuine pyæmia, with abscesses of the lungs, and often recognizable symptoms of thrombosis of the sinuses. Frequently this transmission of the inflammation through the veins is not demonstrable, and yet one finds puriform and broken-down thrombi in the sinuses. The explanation of such cases is this : formation of pus between the bone and the dura, thrombosis by compression, and direct perforation of the sinus wall by the infectious pus. From the point of infection, then, the thrombosis can extend upwards, and cause a purulent meningitis ; and besides this there will be the consecutive disturbances of the circulation in the pia, as well as in the cortex cerebri.

The length of time which elapses between the first formation of pus in the bones and the beginning of the meningeal inflammation is not definite, fixed for all cases. Inflammation sets in generally in the first or second week after the injury; brain symptoms appear sometimes immediately, sometimes after the lapse of weeks. We have seen, indeed, purulent meningitis arise even after fragments of bone had become detached by suppuration. This subject will, however, be more appropriately treated in detail in connection with traumatic meningitis.

Inflammation of the bones of non-traumatic origin also gives rise to meningitis. Apart from those rare cases where scrofulosis seems to be a factor in the affection of the bones of the skull, we refer particularly here to chronic gummy ostitis of syphilitic origin. Notoriously the favorite seats for this form of disease are the frontal and parietal bones; but it can extend over the whole skull. Generally gummy growths are, ab initio, multiple, and if not checked by appropriate treatment, they increase in size, become confluent, bring about atrophy of the bones, and cause sequestra by cutting off intermediate portions of bone from the circulation. A peripheral formation of new bone very often falls a sacrifice also to the new growths.

The ultimate fate of gummata is a variable one. They sometimes become cheesy, sometimes undergo mucous softening, and, even after this latter process, are reabsorbed. Calvaria, riddled like a sieve, are to be seen in every museum. Suppuration of a gummy tumor leads to an external opening; the bone then becomes carious, and portions of bone the full thickness of the skull are sometimes exfoliated. Not uncommonly simultaneous gummy inflammation of the dura complicates the process going on in the bone, and it becomes possible for the inflammation to extend to the pia. The development of a purulent meningitis, as the final stage of the disease, is by no means common under these circumstances—at all events, is less common than meningitis resulting from simple caries of the petrous bone. Under these latter circumstances, as well as under those above mentioned, atmospheric germs seem to play a great part.

Specific processes in bones have been observed in rather unusual situations; for instance, in, or in the neighborhood of, the sella turcica, in the wings of the sphenoid, in the basilar portion of the occipital bone; but for gummy periostitis, in these situations, to lead to purulent basilar meningitis, is a rarity; on the contrary, chronic processes extending to the pia are rather more common—(see above under chronic basilar meningitis).

Caries of the upper cervical vertebræ has been known to give rise to upward progressing inflammation of the pia, even involving the base of the skull.

2. *Otorrhœa* arises from various conditions, which are not all of equal importance in respect of giving rise to meningitis (acute and chronic otitis externa, myringitis, otitis media, polypi of the ear). It arises also from deep-seated inflammation, caries of the wall of the external meatus, caries of the wall of the tympanic cavity, caries of the inner ear [labyrinth]. Purulent inflammation of the soft parts in the meatus and tympanic cavity easily leads to caries of the bone; and, indeed, danger is not confined to those cases in which the process is localized in the interior of the petrous bone, but analogous processes in the bony external meatus, in the mastoid cells, or in the wall of the tympanic cavity, are fully capable of giving rise to purulent inflammation of the pia.

All authorities, particularly Troeltsch, call attention to the smallness of the distance between the superior wall of the external meatus and the dura, to how nearly the transverse sinus and the mastoid cells approach the posterior wall of the tympanic cavity. The floor of this cavity is in the very nearest vicinity of the internal jugular; the internal carotid and the cavernous sinus pass very near the anterior extremity of the cavity. The superior wall of the cavity, too, is only separated from the dura and the superior sinus petrosus by a thin and often defective plate of bone; and besides this, the superior wall contains the petroso-squamous fissure (see Troeltsch, Handbook of Diseases of the Ear). On the inner wall of the cavity of the tympanum are the fenestra rotunda and fenestra ovalis, whose delicate membranes can offer but a trifling resistance to progressive suppuration. From the inner ear [labyrinth], finally, the path along the track of the auditory nerve lies perfectly patent for the extension of the inflammation inwards.

If caries of the petrous bone lead to leptomeningitis, in some cases pachymeningitis forms the intervening step, and this is particularly the case when the inflammation propagates itself through the roof of the tympanic cavity, or by perforation of the posterior wall of the so-called mastoid antrum filled with cheesy pus. Troeltsch considers the former method the more common, and this is not surprising when we consider the frequency of congenital defects in the roof of the tympanic cavity, the liability to perforation which caries brings with it, and finally, the easy means for the transit of the inflammation which is offered by the vessels and their sheaths passing in from the dura through the petroso-squamous fissure. We can scarcely lay sufficient stress on the gravity of inflammation in a small cavity, from which it is difficult for pus to find vent, and which allows access to atmospheric germs from two sides (the Eustachian tube and the perforation of the tympanum).

Extension of the inflammation from the dura to the pia is, in our experience, rather subacute. Already adhesion has taken place between the dura and pia, with appreciable thickening of the former; in some cases, indeed, newly formed vessels have been demonstrated in the adhesions, which vessels manifestly conduct the inflammation from one membrane to the other. It is admitted on all hands, moreover, that pre-existent organized bridges of tissue perform the same service, and that the mere contact of the dura—covered on its inner surface with a thin layer of pus—with the pia, amply suffices for the infection of the latter.

Caries of the inner ear is then by no means absolutely necessary for the occurrence of purulent meningitis, though it is often the sole cause.

Troeltsch and others have found the membrane of the fenestra rotunda destroyed, and also the fenestra ovalis laid bare by detachment of the stapes; and instances are known of the establishment of a communication between the tympanic cavity and the semi-circular canals. Thus can pus find its way to the labyrinth and the cochlea, and thence along the fine branches of the auditory nerve and its sheath to the base of the brain. The same part can apparently be played by the facial nerve in the Fallopian canal after perforation of the walls of the canal. Cases are also on record in which, without any affection of the bony tissue, the inflammation of the tympanic cavity, after perforation of the fenestra rotunda, has extended to the membranous portion of the inner ear, and thence to the auditory nerve. A variety of other ways of extension have been already spoken of under the head of pachymeningitis. (See Moos, Handbook of Diseases of the Ear.)

We must not lose sight of the fact that disease of the brain may ensue on simple otorrhœa, without caries, without direct communication with the cavity of the skull by perforation, but simply with evidences of inflammation of the soft parts. There is a double venous flow in the diploë of the petrous bone, with free anastomosis; the veins empty, some inward into the sinus of the dura, some outward into the veins of the head and of the organ of hearing; the latter, however, lie in the territory of the inflamed soft parts. In this relation Troeltsch correctly perceives a chance for the progress of the inflammation into the skull: thrombosis of the veins of the soft parts, its extension to the veins of the diploë, further extension toward the origin of the sinus; the infectious character which the thrombi acquire in breaking down, gives opportunity for progressive malignant thrombosis of the sinus of the dura, and this, again, leads to thrombosis of the pulmonary artery. But the possibility of meningitis still exists; meningitis can arise secondarily to the thrombosis of the sinus, and have a share in the fatal result.

Polypi.—There are cases on record of meningitis and death from the presence simply of aural polypi which gave rise to caries of the petrous bone. We cannot speak with authority on this head, but it seems more correct to reverse the sequence and regard the polypi as the result of old otitis media.

Foreign bodies.—There are, finally, cases recorded of fatal purulent meningitis from rough and unskillful attempts to dislodge needles and like foreign bodies which have got into the ear.

3. *Puriform softening of a thrombus in the sinus.*—This

can, in its upward progress, give rise to purulent meningitis. In one case the veins, leading from the longitudinal sinus to the pia, were found filled with a chocolate-colored, puriform, softened mass which extended quite into the large veins of the pia. Thus was caused that form of meningitis secondary to caries of the petrous bone, but without direct transmission, of which we have already spoken.

4. *Panophthalmitis.*—Purulent meningitis, beginning unilaterally, has been observed after suppuration of the eyeball (Church). Probably, in such cases, the pus travels along the subpial space of the optic nerve—a view which may be presented, although the anatomical demonstration in this case is lacking.

5. *Erysipelas capitis.*—Some authors doubt that purulent meningitis often follows erysipelas. Our own experience in this matter includes a good number of cases of purulent meningitis which arrange themselves in two classes. The first includes cases of erysipelas, leading to a phlegmon of the scalp, though in only one case did the inflammation have time enough before death to lead to the formation of a collection of pus; osteo-phlebitis of the bones of the skull was found, but the remainder of the course, which the inflammation followed to the pia, was not detected. The second class includes certain cases in which purulent meningitis ensued on erysipelas, attended by the formation of bullae and profuse superficial suppuration of the scalp; in this class, too, the path of the suppuration to the pia is unknown. These observations do not, of course, invalidate the view that severe brain symptoms may be only functional, and stand in no causal connection with the fatal result.

6. *Erysipelas complicated by parotitis.*—In these cases, also, the entire path to the pia, which is pursued by the inflammation, is not known.

7. *Carbuncles of the cheek, upper lip, and neck.*—We have seen a typical case of purulent meningitis follow carbuncle; the thrombosis of the sinus, dependent on osteo-phlebitis, was primary, the meningitis secondary.

8. *Old intra-cerebral affections.*

a. Necrotic deposits in the vicinity of the great ganglia, pouring their contents on the base of the brain through an opening of a larger or smaller size, may give rise to meningitis of moderate intensity. We have ourselves observed perforation of the anterior perforated space downwards, by a semi-fluid collection, of embolic origin, in the head of the corpus striatum.

b. Tumor of the base; sarcomata; cheesy masses. In the vicinity of these, meningitis of no trifling intensity has been found *post-mortem.*

c. Abscess of the brain. The bursting of an abscess of the brain outward, either near the base or near the convexity, gives rise to acute meningitis, spreading rapidly in all directions, its maximum intensity being at the point of rupture. This may be an important episode in the course of an abscess of the brain.

Pathological anatomy.—Has been already partially exhausted above. It is very evident that the anatomical appearances must vary materially, owing to the variability of the etiology; but, in general, the constant appearance is that of more or less extensive infiltration of pus in the pia, especially marked in the track of the larger vessels, and in all degrees of intensity, presenting under the microscope evidences of migration of the colorless corpuscles. The cortex is rarely, if ever, spared by these various forms of meningitis, but presents evidences of a marginal immigration from the pia, as well as of more or less active emigration from the vessels of the cortex and neighboring portions of the gray matter, but without noteworthy changes in the ganglion cells.

In the typical forms of meningitis which originate from the petrous bone, the extent of the morbid changes is sometimes confined to a portion of the base—the pons, one side of the pons and immediately contiguous parts,—sometimes involves the whole base, sometimes the whole base as well as the convexity of that side on which the process originated, sometimes the base and both sides of the convexity. It is easy to see why in the majority of cases the inflammation begins on the base or the temporal lobe; the immediate neighborhood of the roof of the tympanic cavity, of the posterior wall of the petrous bone (the mastoid cells), of the internal auditory meatus, must bear the brunt. The inferior surface of the temporal lobe, the vicinity of the gyrus uncinatus, this body itself, the superior and inferior surfaces of the cerebellum, the vicinity of the corresponding auditory, hypoglossal, and facial nerves, and thence the whole base, are the parts chiefly affected. A not unusual form is limited almost completely to the cerebellum and the base as far as the chiasma. In a case of this kind the pia covering the convexity was but slightly affected; but, on the other hand, the process extended far down the vertebral canal, a condition which is prominent in all these cases. In a large portion of cases the intra-ventricular fluid is increased, which fluid is of a thin, purulent character more commonly here than in those forms of meningitis of which we have already spoken. In correspondence with the increase in the amount of the fluid, the choroid plexuses are often found not only hyperæmic and turgescent, but even infiltrated with pus. The quantity of fluid, however, is never very great, and in some cases is thinly purulent on the side where the inflammation began, but serous on the other.

The course, then, which the inflammation has taken from the petrous bone is in one case easy, in another difficult to determine.

The result of a series of our own observations is as follows:

1. Perforation of the posterior wall of the petrous bone; the opening leads into the mastoid cells, where a number of bits of necrotic bone are imbedded in cheesy pus; separation of the dura from the bone; fetid, purulent infiltration of the former to a considerable extent; adhesion to the pia; widely extended basilar meningitis, reaching down the vertebral canal as far as the third cervical vertebra; thin, purulent fluid in the ventricles; convexity nearly intact.

2. A large perforation of the roof of the left tympanic cavity; separation of the dura from the bone, and perforation of this membrane; adhesion to the pia; extensive meningitis of the base and convexity of the left hemisphere, with slight affection of the right; moderate extravasation in the pia, at the base; moderate serous transudation into the ventricles.

3. Suppurative inflammation of the internal auditory canal; auditory and facial nerves sheathed in pus; moderate formation of pus in the vicinity of the right auditory and facial; inflammation less marked on left side; a corresponding distribution over the rest of the base; moderate affection of the convexity; no hydrocephalus; cheesy masses in the petrous bone.

4. Perforation of the superior wall of the external meatus; caries of the bone; no separation of the dura, but pachymeningitis purulenta interna; adhesion to the pia; meningitis of both convexity and base, of moderate extent; no hydrocephalus.

For further details, consult the monographs of aurists.

Very marked changes are often found in the auditory and facial nerves in those cases in which the inflammation has extended along them, varying in degree from purulent sheathing, discoloration, and thinning, even to complete softening and disorganization of the nerves. Of great importance in this connection are observations such as that of Martini. In a case of this character he found that pus had made its way along the nerve to the basilar artery, around and in the wall of which was a small abscess. The abscess, as well as the adjoining tissue, contained large nests of micrococci. Martini is inclined to attribute to them the progressive suppuration. Such facts cannot surprise us when we regard the ease with which germs can gain access to the petrous bone, standing as it does in communication with the atmosphere.

Conditions which favor high blood-pressure in the brain come here but seldom into play, and hence observers have often remarked on the discrepancy between the anatomical appearances and the symptoms during life. Sopor and coma constitute the often rapidly advancing close of the scene. There are cases, however, in which coma and sopor are postponed to the very last days of life.

If the meningeal inflammation ensue on thrombosis of the sinus, the latter complicates the anatomical appearances.

In one case the thrombus extended upward into the commencement of the longitudinal sinus and a portion of the veins of the pia which empty into it. The thrombi were breaking down into fetid, purulent matter. The meningitis was seated on both sides of the convexity, the base being relatively free.

Moreover, under these conditions the meningitis can begin at the base—the purulent pachymeningitis, which has taken hold of the sinus, spreading to the cerebral surface of the dura, and thence to the pia.

An abscess of the brain can break through to the convexity by a small opening as well as by a large one.

Meningitis consequent on facial erysipelas or carbuncle has been already briefly alluded to. As to the latter, it is to be remarked that the path to the interior of the skull is generally through the orbit. In one case the parts external to the eyeball played the chief part; for, besides decided stasis in the retina, nothing abnormal in the eyeball was detected during life. Before death intense chemosis conjunctivæ took place.

Some cases, which occurred in the course of diphtheritis, were obscure.

Among other forms of diphtheritis associated with meningitis, Politzer observed it once with conjunctival diphtheritis, though in his case an intermediate pyæmic infection seems to have been a factor. We ourselves have known rapidly fatal meningitis to complicate diphtheria of the fauces; but we shall return to these cases in a later section.

Incidental Causes.—These are various. Confinement of pus within the petrous bone is often spoken of, and in former times the cessation of a discharge was considered always highly unfavorable. The fact cannot be denied, and is confirmed by manifold experience, that when a discharge suddenly ceases, the danger is great lest suddenly increased pressure rupture a delicate partition wall or a thin membrane, and mechanically favor the progress of pus. But it is not to be inferred, on the other hand, that there is no danger, provided the discharge continue. It is a matter of *à priori* reasoning, and confirmed by experience, that meningitis can begin at any time; though, indeed, it is more likely to occur when for any reason the free discharge of pus is interfered with. Such reasons are: swelling and narrowing of the external meatus, with perforation of the membrana tympani; large polypi, which close up the opening in the membrana tympani and fill the meatus; a perforation of insufficient size, which is closed by swelling; confinement of pus within the mastoid cells, the opening of communication [with the middle ear] being closed by inspissated pus; or again, a tympanic cavity, replete with pus, may prevent the evacuation of the mastoid cells. If, simultaneously, the membrana tympani be intact and the Eustachian tube closed—a common thing—the pus is completely pent up; and if the drum membrane be unusually tough, a rupture, which under other circumstances would take place, may fail to do so; or the mastoid cells may be prevented from emptying themselves by an adhesion between the drum membrane and the promontory.

Now and then meningitis appears, not as a suddenly intercurrent event in the course of a chronic affection of the ear, but as following immediately upon an acute exacerbation of the latter. It is easy then to recognize acute trouble in the ear by the sharp pain and the general indications of inflammatory action; but its extent, and whether the meninges are involved or not, remains doubtful until symptoms of meningitis appear. The interval is generally a short one.

As other incidental causes may be enumerated: falls and blows on the head, caries of the petrous bone being already present (Toynbee and Griesinger); errors in diet (Schwartze); too forcible injections into the ear (Troeltsch); gangrene of the soft parts of the ear (Grossmann); thorough wetting and exposure to cold, with severe muscular exertion (our own observation). Finally, cases occur without assignable cause.

Symptoms.—The degree of acuteness varies very greatly, according to the causation, and it is therefore impossible to include all cases under one description. This compels us to select a type, and as such we shall make use of the form of meningitis which starts from the petrous bone, and which still varies within wide limits.

Let us begin with the report of a case:

26. A laborer, aged twenty-seven; entered hospital April 29th, and died May 12th. Cause of death of father unknown; mother alive and well; no history of sickness in childhood. A slight wound of the face at six years of age; a fracture of the right arm at twelve; otherwise, till 1868, perfect health. Since that time, persistent fatigue and feeling of tension (spannende Gefühle) in legs; loss of flesh and muscular power, especially in legs, but up to this time no interference with locomotion. Since 1870, thirst, impelling him to drink water freely, but appetite for food not increased. A sister died of diabetes, and patient affirms that he labors under the same disease. No symptoms of importance connected with stomach or intestines. The quantity of urine was in direct proportion to the amount of fluid ingested, and estimated by him at eight pints daily. Thinks that his face has become thinner within six weeks. Has been a hard drinker. In last eight weeks has suffered from attacks of severe headache, beginning over the right eye, and extending obliquely across the head to the left ear. For four weeks almost complete deafness on left side, and profuse otorrhœa attended with severe pain.

While this history was being taken, the patient exhibited a sudden disturbance of speech, made no progress in what he was saying, repeated the same word at least ten times, and could not say the following word; after some time a couple of correct words followed, and then his speech degenerated into an unmeaning conglomeration of sounds. *The patient became aphasic in the course of a few minutes.* Temperature 38.4° (101° F.).

April 30th.—Psychical disturbance, confusion, wandering; does not know where he is or what is happening to him. *Aphasia.* A large amount of sugar in his urine; volume, 2800 c.cm.; specific gravity, 1042. A quiet night; *slight paralysis of the left facial nerve since yesterday evening.* Temperature 37–38.4° (98.8°–101° F.). Pulse 116.

May 1st.—Night quiet. In morning grinding of teeth, wandering, lack of perception, thickness of utterance, and *aphasia.* Paralysis of left facial; moderate discharge from left ear, and complete deafness on that side. Temperature 37.6° (99.8° F.). Pulse 108. Temperature 38° (100.5° F.). Pulse 120. Quantity of urine not ascertained; specific gravity, 1038.

May 2d.—Night quiet. Wandering and delirium increased; *aphasia.* Gait normal; no paralysis of extremities. Facial paralysis as before. Temperature 38.4° (101° F.). Pulse 120. Temperature 38° (100.5° F.). Pulse 104. Headache; groans frequently; no real sopor. Passes his water in bed. Ophthalmoscopic examination by Prof. Horner. Refracting media normal; papillæ redder than normal; contour not sharply defined; veins unduly dilated; some swelling of the papillæ; commencing choked-disk.

May 3d.—Condition the same. Temperature 37.4° (99.4° F.). Pulse 104. Temperature 39° (102.3° F.). Pulse 124. Vomited twice in evening. Urine, V. 3000; specific gravity, 1040.

May 4th.—Urine, V. 1180; specific gravity, 1040; 87.5 grammes of sugar in twenty-four hours; 87 grammes urea (3000). Old diabetes. An examination of the ear reveals unquestionable perforation of the membrana tympani; and, with probability bordering on certainty, caries of the petrous bone; hearing on left side zero. Mastoid process normal, not tender on pressure.

Wandering; delirium; lack of perception of place and time; severe headache, of which the objective signs are present; groans, and grinds his teeth frequently. Retention of urine. Choked-disk moderate in degree, but more distinct. Vomiting. Temperature 39.8° (103.7° F.). Pulse 116. Temperature 37.8° (100° F.). Pulse 116. Aphasia less marked; pupils alike, respond badly to light; muscles of eye normal; no ptosis; tongue protruded in median line, with a white coat; left facial paralysis rather less marked. Patient can stand and walk, though unsteadily; no paralysis of extremities. Sensorium normal; no hyper- or anæsthesia; heart and lungs normal; no hypertrophy of liver or kidneys.

Diagnosis.—Diabetes; commencing meningitis on the left side, arising from an affection of the petrous bone, which is also the cause of the aphasia. Facial paralysis from an affection of the facial nerve, either in the Fallopian canal or in the internal auditory canal.

May 5th.—Temperature 37.8° (100° F.). Pulse 116. Temperature 38.2° (100.8° F.). Pulse 124. Urine, V. 1450; specific gravity 1038; urea 45.24; sugar 76.27; albumen. A quiet night. A helpless, stupid, and rigid appearance; rigidity of the muscles of the neck, not of the extremities; no convulsions; sensorium relatively normal; no sopor, though lack of realization of his situation; power of speech has returned; aphasia has disappeared; can answer simple questions correctly; knees give way when he attempts to stand; tongue with thick white coat; facial paralysis more marked; vomiting; persistent headache.

May 6th.—Temperature 38° (100.2° F.). Pulse 120. Temperature 38.2° (100.8° F.). Pulse 152. Urine, V. 1720; specific gravity 1035; urea 48.6; sugar 83.4; albumen. Condition unchanged; no increase of mental symptoms; aphasia has vanished.

May 7th.—Temperature 37.8° (100° F.). Pulse 116. Temperature 37° (98.8° F.). Pulse 140. Urine, V. 1900; specific gravity 1032; urea 52.44; sugar 91.7; albumen. Restless night; fell out of bed; sang and yelled; lies with closed eyes; sings; attention can be attracted only for a moment and with difficulty; answers unintelligently; no return of aphasia; pupils unsymmetrical (old synechiæ and atrophia); headache; no paralysis of extremities; no contractures, but rigidity of the neck.

May 8th.—Restless night; sang and yelled; now lies quietly in bed; delirious; does not move; resists passive movements; cries and groans; hyperæsthesia of skin, periosteum, and bones; rigidity of neck; when let alone sinks into sopor, but starts at the least irritation; answers unintelligently; no aphasia; no ptosis; muscles of the face normal; left facial paralysis; no paralysis of hypoglossus; abdomen retracted (for the first time); abdominal muscles tender on pressure; headache; no convulsions or symptoms of thrombosis of the sinus.

Diagnosis.—Purulent meningitis, more marked on left side. Temperature 37.4° (99.4° F.). Pulse 136. Temperature 36.6° (98° F.). Pulse 136. Urine, V. 2250; specific gravity 1032; urea 49.5; sugar 92.02; albumen, but no sediment or casts.

May 9th.—Temperature 37° (98.8° F.). Pulse 132. Temperature 37° (98.8° F.). Pulse 120. Urine, V. 1150; specific gravity 1026; urea 31.16; sugar 49.4; albumen; no casts. Restless night; delirious; noisy; urine and fæces passed in bed; rigidity and tenderness of neck increased; otherwise as at last report, except that the right angle of the mouth moves more feebly than hitherto; bilateral choked-disk, but *redness and opacity more marked* in left (Horner).

May 10th.—Temperature 38.2° (100.8° F.). Pulse 132. Temperature 37.8° (100.° F.). Pulse 152. Urine, V. 950; specific gravity 1025; urea 28.31; sugar 34.2; albumen. Delirious in night; this A.M. sopor alternating with moments of relative intelligence; indications of headache; groaning; ptosis; pupils and condition of retina as at last report; muscles of the ball normal; again an increase in the facial paralysis; grinds his teeth, and passes his water in bed.

May 11th.—Temperature 38.8° (100.° F.). Pulse 152. Temperature 36° (96.9° F.). Pulse 124. Urine not examined. Restless night; unconsciousness nearly complete; vomiting; automatic movements of extremities and tongue; rigidity of neck; urine passed in bed; other symptoms the same.

May 12th.—Temperature 36.2° (97.2° F.). Pulse 136. Temperature 37.4° (99.3° F.). Pulse 124. Urine ? Delirium; rigidity of neck; rolling of eyeballs upward; no strabismus; facial paralysis, and pupils as at last report; no convulsions. In the evening became deeply comatose, with irregular pulse and respiration. Death. Duration eleven days.

Autopsy.—Calvarium thin; dura somewhat injected; in the longitudinal sinus fluid blood; but little serum in the subarachnoid space; some injection of the large and medium-sized vessels of the posterior two-thirds of the brain; pia of the frontal lobe pale. A large quantity of serum mixed with pus flows from the vertebral canal; the subarachnoid space on the base surrounding the chiasma much infiltrated with pus, as also the inferior surface of the occipital lobe; no miliary tubercles; moderate dilatation of the left lateral ventricle, which contains a thin purulent fluid with some coagula; right ventricle the same; consistency of brain good; moist and pale on the cut surface; transverse section of vessels but slightly marked; a few small points of ecchymosis.

The dura in the vicinity of the internal auditory canal, in the diameter of about one cm., is covered by a grayish red, easily detachable tissue, which completely surrounds the facial and auditory nerves. In separating the dura from the pyramid of the petrous bone, which is done with relative ease, there is found on the posterior wall of the petrous bone, and immediately alongside of the transverse sinus, a mass of thick pus, the size of half a cherry. On removal of this an opening as large as a bean is seen in the bone, detached in which opening lies a bit of porous and necrotic bone, nearly filling the opening. In the superior wall of the petrous bone is also a hole as large as a pea, and at its base a little pus. The mucous membrane of the mastoid cells is much swelled and injected; there is a quantity of pus in the middle ear, and a portion of the auditory nerve is much thickened by a gelatinous tissue. Spleen healthy.

As the sequence of events and the development, so does the duration of these affections vary within wide limits, depending, as it does, on the degree of acuteness with which the process spreads to the pia. In general one can divide these cases into three classes, which will as a rule suffice.

a. Cases of from twenty-four to forty-eight hours' duration, rapidly fatal, in which, however, sometimes trifling symptoms, such as headache, dizziness, a little fever, all pointing to an intracerebral affection, had preceded. These prodromata indicate rather a chronic process of the dura, or even of the pia and cortex—such as pachymeningitis, with marked thickening of the membrane, adhesion by connective tissue to the pia; in one case the formation of a tough mass of connective tissue, strongly adherent to the cortex and extending into its substance in the shape of a strongly marked thickening of a vascular sheath, was noted.

b. Cases of two, three, or four days' duration, of an extremely acute commencement and course, rapidly leading to brain symptoms and ending with sopor, coma, sometimes convulsions, and death. Here we generally find a long brewing, but, at the last, sudden perforation of the dura of large size; no protecting connective tissue formation between the dura and pia, and in the pia itself, having taken place. The meningitis is of great extent, generally double, and never spares the convexity.

c. Cases of from four to fourteen days or three weeks. The case above reported belongs to this class. The gradual sequence of the symptoms in these cases is remarkable; the fever is slight, *the sensorium remains long intact*, the symptoms are slight and incomplete, leading one to hesitate in the diagnosis. The convexity is never much involved, the inflammation of the pia is chiefly marked at the base, the hydrocephalus slight. The above-reported case is the only one in which we have observed aphasia.

The *commencement* of the affection is, in many cases, marked by prominent symptoms, in other cases not. In many cases a single or repeated chills have been observed; in other cases high fever, without an antecedent chill, ushers in the brain symptoms. The general rule, however, is for the brain symptoms to precede, and the high fever to follow.

The first brain symptom is always *headache*, sometimes extending over the whole head, sometimes limited to the vicinity of the diseased ear, sometimes shooting from one ear to the other, sometimes moderate, sometimes of unbearable intensity, sometimes periodically increasing and decreasing, sometimes always of like intensity. If the local trouble in the ear was attended by pain, increase in the extent and severity of the pain marks the commencement. Then comes *giddiness*, though we do not find it noted in every case; and associated with the giddiness is generally *vomiting*, or at any rate nausea. The patient complains of noises in his head, general painful sensations, and that his senses are leaving him. These signs may be developed very gradually, but their bearing is none the less marked on that account; often the otorrhœa had been attended for a long time by headache, not very acute—depending, as will presently appear, on chronic changes in the dura mater and petrous bone.

Gradually more important symptoms appear: increase of pain, fever, chilly sensations; this stage may continue for some time, and the patient get a sudden access of most intense pain in the head and become immediately delirious, or, at all events, wander and be unable to concentrate his attention. These symptoms may pass away and return again two or three times before serious danger shows itself. Finally, it may happen that an increase in the local inflammatory action in the ear, with its fever and pain, coincides exactly with the commencement of the brain symptoms, so that the beginning of the meningitis is completely masked. Further, giddiness and vomiting have been known to be the very first symptoms; in some cases *facial paresis* was the first sign, referrible to the canal of Fallopius. In the above reported case moderate headache was the first symptom, then came on a sudden *difficulty of speech*—at first loss of the power of articulation, later *aphasia*. Rigidity of the muscles of the neck and back has been also mentioned as the initial symptom.

The subsequent course, in some cases, keeps the beaten track of meningeal inflammation, *i.e.*, the stage of depression, when symptoms of torpor predominate, follows a longer or shorter period of time during which the prominent symptoms are those of irritation. Then ensues, *though not invariably*, more or less compression of the brain, and finally death, attended by gradual paralysis of the centres of the medulla oblongata. In other cases there is an ebb and flow, as it were; symptoms of irritation alternate repeatedly with those of depression, and death takes place suddenly and unexpectedly, without a single indication of compression having appeared. The autopsy, even, reveals in some cases no sufficient cause of death appreciable by us, so that one is drawn towards the theory of infection which Billroth maintains.

This variable course of the disease, scarcely alike in two cases, is the cause of great variation in the *mental symptoms*. Excitement, jactitation, restlessness, confusion of ideas, are generally only transitory, appear usually toward evening as the temperature rises, and remit in the morning, when an occasional rational answer may be obtained, though the intelligence is not perfect on all points. With manifold variations there comes deeper sopor, out of which the patient can be aroused with relative ease, and, finally, sopor gives place to coma, or else, after a short stage of excitement (delirium, etc.), consciousness is lost once for all, and, reflex action alone remaining, life gradually ebbs away. Genuine symptoms of compression, viz., dilated pupils, non-respondent to light, slow pulse, slow and irregular respiration, marked evidences in the retina of pressure of fluid out of the subdural space into the sheath of the optic nerve, may be superadded to this suppression of function of the cortex, but are not necessary to a fatal result.

The species and form of delirium are extremely variable, and do not require exhaustive description.

Important symptoms connected with the *power of motion* are generally present.

Facial paralyses are common on the same side as the ear affection, and generally the lesion is situated in the Fallopian or the internal auditory canal. The paralysis may have also the characteristic signs of peripheral facial paralysis, be complete, and attended by the characteristic defective reaction to the constant and induced currents. The paralysis may be partial only, and its origin still be referrible to the Fallopian canal: it depends entirely on the precise character of the local nerve lesion. If facial palsy of the same side appear in the course of the brain affection, it may be complete as well as partial, and cases occur in which, owing to the acuteness and rapidity of their course, the nerve-lesion cannot be localized during life. Partial facial palsy of the same side may also be a symptom of meningitis near the entrance of the facial nerve into the medulla, and in some cases we find the Fallopian and internal auditory canals perfectly normal, the cause of the meningitis lying in a perforation from the mastoid cells, or through the roof of the tympanic cavity. In a case under our own observation, both facials were paralyzed one after the other, the cause being extensive basilar meningitis; this was also present in a slight degree in the case which we have reported above.

The hypoglossus of the same side is often simultaneously affected, as evidenced by obliquity of the tongue, the tip being directed toward the diseased ear.

If the meningitis, as is usual, extend forward on the base, the abducens and oculo-motorius become involved. If the inflammation ascends on the back of the peduncle to the corpora quadrigemina, various abnormal contractions in the muscles of the eyeballs are brought about; at the same time paralysis of the abducens and oculo-motorius appears, so that convergence or divergence of the eyeballs is associated with the abnormal contractions. If one oculo-motorius be completely paralyzed, which happens but seldom, the corresponding eyeball is drawn toward the outer corner of the orbit, with ptosis and dilated reactionless pupil.

The condition of the pupils is very variable, often changing many times in the same patient during the course of the disease. At first they are generally contracted and react feebly, either bilaterally, or in some cases *only on the side on which the meningitis originated;* the later manifestations follow the law that, in default of a decided effusion into the ventricles, the dilatation is unilateral, and the branches which go to the pupil share in the affection of the oculo-motorius—yes, and may exclusively be affected. When bilateral weakness of innervation of the oculo-motorius exists, the condition of the pupil varies from time to time, and may at the same time be dissimilar on the two sides. Paralysis of the extremities is very rare.

The same lack of co-ordination in complicated movements, which we have touched on already in connection with similar affections, holds good here: an unsteady, rolling gait, dependent on giddiness, which is often very great; in one case inability to perform delicate manipulations, on account of constant simultaneous movement of other muscles, was noted; that aphasia may occur and aid in the diagnosis, is shown by the above case, in which the affection was on the left side.

Evidences of motor irritation are numerous. Such are:

Rigidity of the neck, a symptom which is almost constant and often very marked and permanent, and causes the patient to bury his head in the pillow. A series of observations led us to refer this symptom to meningitis of the cervical portion of the cord.

Convulsions in the territory supplied by the facial nerve of the affected side.

Trismus and grinding the teeth.

Convulsions in both arms (Troeltsch).

Convulsions in the arm opposite to the diseased ear (Schwartze).

Convulsions in one arm and both legs (*our own observation*).

As for sensation, the often observed and in the above case noted, marked hyperæsthesia of the skin, joints, bones, and soft parts, is especially remarkable. Every movement is painful, locomotion well-nigh unbearable.

Headache in all degrees of intensity is always present during the whole course.

The gastric and intestinal symptoms are the usual ones. Vomiting is rarely absent, constipation in varying degree is present. Retraction of the abdomen and tenderness of the abdominal muscles are very common, but not constant.

Temperature. In acute cases it is generally very high, remits slightly in the morning, rises generally in the second half of the day ; sometimes remains at its height till death, sometimes has a marked fall immediately before death. But in cases which run a slow course, as the above, the temperature is often low, sometimes even lower than normal, and runs up without manifest cause. In cases where compression of the brain has not occurred in the first days, the pulse follows pretty closely the temperature, but is greatly influenced by the subjective condition of the patient.

In the second half of the disease, unless symptoms of compression supervene, the pulse rises, whatever be the temperature, and remains rapid till death, even when the temperature varies greatly. If compression supervene, the pulse may become slow, but never remains so long.

In almost all the cases which came under our observation, albumen was found in the urine, but no indications of deep-seated disease of the kidneys. When the amount of urea was observed, it was always found decidedly increased. The enormous amount of urea excreted in the above case of diabetes is noteworthy.

Opthalmoscopic examination reveals about the same state of things as we have already sketched out under tubercular meningitis. It is to be borne in mind that during the course of the disease evidences of choked-disc will be more marked on the side where the affection originated (Horner).

The lack of agreement which there often is between the condition of things as shown by the autopsy and the manifestations of the disease during life, is striking, and this remark is equally applicable to other forms of meningitis. Frequently death occurs, after a short period of sopor, from paralysis of the medulla oblongata, and the autopsy reveals an extent of meningeal trouble *which surely would not have brought about so early a fatal result, if connected with one of the simple forms of idiopathic meningitis of the convexity.* The fatal result must be dependent on circumstances of which as yet we have no knowledge. We get a valuable hint, however, from the conclusions of Martini, which suggest infection of the blood by products of decomposition. Many authors, but especially Billroth, have dwelt upon the rapid close of those cases of meningitis which run their course under the influence of the access of atmospheric air.

Meningitis associated with erysipelas presents nothing especially remarkable, but is often very difficult of diagnosis. We have to decide whether we have meningitis, a simple anomaly of circulation, or a merely functional disturbance of the brain, dependent on the fever and the poisoned condition of the blood. The difficulties are such that, after much experience, we make a positive diagnosis of meningitis only when either partial or general convulsions, rigidity of the neck, and vomiting occur, and, at the same time, the pulse and temperature, the condition of the sensorium, and the results of opthalmoscopic examination correspond with the supposition of an inflammatory affection of the surface of the brain. The psychical symptoms alone are not sufficient for diagnosis, not even in conjunction with the febrile symptoms—indeed, they are not dependent on a common cause. We have become extremely skeptical in laying much stress on inequality of the pupils and commencing venous congestion of the retina, since we have seen cases in which the latter symptom was present, and at the autopsy *we found no purulent meningitis, but microscopic changes in the cortex, especially migration* *in moderate degree.* If one follow these principles which we have laid down, one may now and then fail to diagnosticate commencing purulent meningitis supervening on erysipelas; but, on the other hand, one will not diagnosticate it when it does not exist.

There are three affections with which meningitis from caries of the petrous bone may be confounded:

1. *Tubercular meningitis.*—Caries of the petrous bone being eminently a scrofulous affection, in the course of an otorrhœa tubercular meningitis may break out entirely independently of the ear affection. The difficulty in making a diagnosis may be appreciated if one considers the extremely variable course of the two diseases; and, before deciding in favor of simple meningitis, there must be valid reasons for connecting it with the affection of the bone.

2. *Thrombosis of the sinus.*—The possible difficulty in diagnosis disappears when one recognizes the fact that by no means all cases of thrombosis of the sinus present during their course characteristic symptoms pointing to the vessels; again, the two affections may be combined; and, finally, thrombosis of the sinus may give rise to marked congestion of the veins of the pia, and consequently to meningeal apoplexies. The chief signs of thrombosis of the sinus are the following:

a. Headache in all degrees of intensity, but which may be very moderate (Griesinger).

b. Secondary thrombosis of the jugular vein. This can be felt on palpation, and gives rise to pain along the course of the vein, and œdema of that side of the neck.

c. Circumscribed œdema behind the affected ear, in the vicinity of the mastoid cells (Griesinger). This is rare.

d. Unequal filling of the jugulars; collapse of the internal jugular, no blood flowing into it from above; and, in consequence of this, easier evacuation of the external into the internal jugular, and diminished fullness of the former. Also rare.

e. General indications of irritation, followed by those of compression, as coagulation progresses upward in the sinus.

f. Evidences in the retina; symptoms of passive congestion by reason of general increase of intracranial tension.

g. Symptoms of pyæmic infection, chills, affections of the joints, abscesses of the lungs, infarctions in the liver, kidneys, and spleen.

The diagnosis of cases in which these symptoms are well marked is easy, but these cases form a small minority, and the evidences of pyæmia are often entirely wanting—often but slightly marked and obscure. Chills appearing late in the course of the disease seem to constitute the most reliable evidence of pyæmic infection; but that they are not pathognomonic is shown by an observation of Schwartze, who noted them in simple meningitis.

3. *Abscess of the brain.*—The differential diagnosis from meningitis will be spoken of hereafter.

Prognosis.

The prognosis of acute purulent meningitis, dependent on disease of the bone, is absolutely unfavorable. It is true there are observations which go to show that meningitis of very slight intensity or short duration—the commencement of the process which is generally found at the autopsy—may be supported and the symptoms disappear. In itself this does not seem very improbable. But, since, in the vast majority of cases (see traumatic meningitis) of purulent infection of the pia, especially when the pus contains already products of decay, the inflammatory signs are progressive, the prognosis of these cases must, at the best, be extremely bad. The prognosis of simple thrombosis of the sinus, even of abscess of the brain, is more favorable, since in both affections the anatomical arrangement of the structures involved is such that nature *may* effect a cure. Cases of recovery from both affections are reported: from thrombosis of the sinus, by means of the gradual establishment of a collateral circulation and mastery of the pyæmic infection; from abscess of the brain, by means of evacuation outwards (Canstatt, Griesinger, Wilde, Sédillot, Lallemand, Toynbee, Moos.)

Treatment.

Is chiefly prophylactic, for, whenever unmistakable symptoms of meningitis are present, our endeavors are fruitless, except in so far as they are directed merely to the symptoms. But it is not without good reason that aurists universally call attention to the necessity of the careful treatment of every otorrhœa ; even if hearing is not improved, such a direction will surely be given to the disease by appropriate treatment as to give nature a chance to effect a cure, and to greatly diminish the danger of the inflammation penetrating into the interior of the skull.

To attain this object the chief means are the following:

1. Careful application of astringents to the inflamed surfaces in the ear.

2. Distention and cleansing of the tympanic cavity by Politzer's method, and subsequent application of astringents.

3. Treatment of the granulations and secondary polypi.

4. General treatment directed against scrofulosis or anæmia, whether the result of a bad constitution or of long-continued suppuration. Special directions for carrying out these recommendations belong within the scope of works on diseases of the ear.

As soon as a brain complication appears, or symptoms which are even suspicious, all means at hand which avail against intracerebral inflammation are to be immediately resorted to. There is reason to hope that a commencing inflammation of the pia may be checked, if the anatomical conditions are not especially unfavorable.

V. *Metastatic Meningitis.*

We come now to consider certain forms of meningitis which arise as terminal complications in the course of acute diseases of another nature, and it seems advisable to retain the term "metastatic," although the views under which it originated are long since obsolete. We consider it a tenable view, that in nearly all the cases which come under this category we have to deal with an infection of the pia, which has a certain analogy with the infection from contiguity, of which we treated in a former section. In the latter the path traversed by the infection is a short one; in the former, however, long, and, in the existing state of knowledge, is accurately ascertained in only a small proportion of cases. Careful anatomical investigations now and then give us a hint as to the path taken by the infection. If it be shown with certainty, in a case which will presently be reported in short, that an infectious embolus from the heart gave rise to partial basilar meningitis, which needed but the one element of time to become general, it does not seem far-fetched to suppose that in other cases of "metastatic" meningitis also, it is through the arterial current that the infectious matter gets into the tissue of the pia. Similar experiences and considerations apply to the origin of certain cases of brain abscess. In view of the extreme facility with which suppuration extends from a limited over a large area of the pia, there is nothing incredible in such a supposition.

The form of meningitis of which we speak occurs in the following connections:

1. *Croupous pneumonia.*—A meningeal complication in pneumonia is by no means rare, but we must distinguish between two sequences of events. In places where cerebro-spinal meningitis has been present as an epidemic, it has been observed that pneumonia, with striking frequency, complicates this form of inflammation. On the other hand, meningitis of the convexity can ensue on pneumonia without epidemic influences entering into the case in any way. We have seen a large number of these accessory or terminal meningeal inflammations here in Zürich, and hence must dissent from those who characterize them as rare. Chvotek, in Vienna, in 220 cases of pneumonia, found meningitis four times; in Zürich the percentage is higher. We have never seen the combination with epidemic cerebro-spinal meningitis.

Immermann and Heller noted during the prevalence of epidemic meningitis this complication in nine out of thirty cases of pneumonia, from which it appears that pneumonia decidedly increases the disposition to cerebro-spinal meningitis. (See Juergensen on Pneumonia, who also saw some cases of this kind.)

If the question be asked whether there is anything peculiar and characteristic about pneumonia occurring in connection with non-epidemic meningitis, we would answer that in the majority of cases the pneumonia is in the stage of purulent infiltration; in the minority, in that of red hepatization. However tempting it may be to state that the former condition is *always* found, facts do not warrant us in so doing. But, in two of the cases in which the pneumonia was found in the stage of suppuration, friable thrombi were found in the pulmonary veins—indeed, in one case, even the thrombi were partially broken down by suppuration. The supposition seems warrantable that puriform broken-down material gets into the arterial current, is carried by it to the pia, and there sets up purulent inflammation. We venture to propound this explanation, but do not wish to develop it further. It is chiefly feeble and decrepit subjects, especially hard drinkers, who fall victims to this combination of pneumonia and meningitis of the convexity.

2. *Pneumonia with fungoid vegetations.*—It is notorious that if the action of the heart be very feeble, coagula are often formed in the heart, which coagula, if the patient live long enough, are transformed into what are known as fungoid vegetations. These vegetations, after the resolution of the pneumonia, or during its resolution if long delayed, may be partially separated from the wall, and their softened contents mingle with the blood of the pulmonary artery; or else, bits of the vegetations or secondary coagula may be carried into the lungs, and give rise to hemorrhagic infarctions. We have observed this twice: in one case the infarction in the lung was relatively fresh, and the pia quite normal; in the second, the infarction had undergone puriform softening, and the result was intense meningitis of both base and convexity. Both cases were marked by chills, and in both ulcerative endocarditis was diagnosticated. In these cases one is tempted to attribute the meningitis to embolic transmission, but the chain of evidence is not complete.

3. *Ulcerative endocarditis.*—Meningitis is rarely combined with this affection, but Heschl describes a series of cases. Their origin seems to differ greatly. In some of the cases thrombosis of the sinus, which had undergone puriform softening, seems to be the cause; in others the meningitis seems to depend on embolic processes which have not yet been clearly demonstrated. Small masses of necrotic tissue, and small hemorrhagic infarc-

tions of the cortex and white substance bordering upon it, are nevertheless more common than meningitis.

We have known partial basilar meningitis to originate where the artery of the fissure of Sylvius is given off from the right internal carotid. The commencement of the artery was plugged by an embolus from an endocarditic heart, and the embolus had also, as usual, given origin to a patch of necrosis in the depths of the right group of great ganglia. The infectious (mycotic) character of the embolus was shown by Eberth. The infection of the pia had taken place *through the wall of the vessel*, and developed mechanical as well as dynamical effects.

In a similar case reported by Wilks, the embolus did not infect the pia, but the patch of necrosis resulted in a genuine abscess of the brain. Another interesting case has been reported in which ulcerative endocarditis of the *right* side of the heart led to foci of suppuration in the lungs; besides this there was basilar meningitis, probably originating from the lungs (Dickinson).

4. *Pyæmia.*—Meningitis rarely complicates pyæmia, which is more frequently associated with secondary inflammations in other organs. If we examine the present condition of the pyæmia question, thus much is clearly projected from the ever-changing confusion of views: that it is a definite biological organism which, to say the least, accompanies the manifold primary and secondary manifestations. It can no longer be doubted that the poisonous character of the pus, as well as the general infection, depends on the presence of organized elements. From suppurating open surfaces these can penetrate the tissue, break it down, and get into the blood, without doubt, by passing through the vascular walls, and, by being carried into distant organs, can excite suppuration and inflammation in them. They are certainly not second in importance to the long-known means of infection, thrombi and macroscopic emboli.

Pyæmic meningitis occurs in connection with pyæmic abscesses in the lungs; in this case the supposition seems justifiable, that in correspondence with the late appearance of the meningitis, after the infectious thrombosis of the pulmonary veins had existed a certain length of time, the poisonous material was carried farther by the arterial current. But pyæmic meningitis has been known to occur *without simultaneous abscesses in the lungs*, if indeed we can place much confidence in a number of very conflicting and incomplete reports of autopsies. The genesis of these cases is obscure, but the possibility that infectious particles pass through the pulmonary capillary circulation is suggested to us.

5. *Acute rheumatism.*—Meningitis occurring in the course of rheumatism is not in every case to be regarded as itself of rheumatic nature. Some of the cases which have been reported were really cases of cerebro-spinal meningitis, with intense peripheral tenderness; but however conscientiously one may criticise, there always remains a number of cases in which meningitis of the convexity certainly appeared during the course of rheumatism, without the intervention of either endocardium or pericardium as connecting links (Stoll, Scudamore, Niemeyer, Lebert). The reports of Rigler, as to the great frequency with which these two affections are associated in Turkey, are interesting. The connection of meningitis with rheumatism is threefold:

a. Endocarditis is the connecting link, so that the combination is: rheumatism, ulcerative endocarditis, meningitis.

b. Purulent inflammations of the serous membranes form the connecting link, endocarditis being present or not, as may be. In this case purulent meningitis is secondary to purulent inflammation of the serous membranes; this is very rare, and the exact connection is unknown (an observation of our own, in which the coincidence was striking, but the manner of transmission obscure).

c. Meningitis complicates rheumatism without there being any purulent deposits in the body, or any affection of heart; the connection here is also obscure.

6. *Purulent pleuritis and pericarditis.*—The same laws apply here. While we may assume that pus is absorbed into the system and circulates with the blood stream, no one has yet actually proved this in any individual case.

7. *Cheesy deposits in the lungs.*—Meningitis can complicate this condition, and miliary tuberculosis—which one would much

sooner expect—have no share in it. There are two ways in which this comes about:

a. Meningitis is present; in the lungs no miliary tubercles, but masses of cheesy inflammatory products, which might, indeed, have given rise to a fresh outbreak of miliary tubercles. The meninges are free from tubercles, but present, microscopically, widely extended, though not very intense, suppuration; no hydrocephalus; the plexus normal.

b. Besides chronic changes, miliary tubercles are found in the lungs and pleura, and meningitis in addition; *but the inflammation of the pia is a simple purulent one, not a tubercular inflammation.* Many such cases have been observed. The meningitis resembles that just described under *a*, and, of course, cannot be classed with the tubercular forms.

We take this opportunity to mention still another class of cases, to which, on account of their uncertain position, we have as yet not been able to call attention. Hitherto *many cases of simple hydrocephalus, associated with general miliary tuberculosis, have been described*, the pia showing evidences of inflammation, *but no miliary tubercles.* Without wishing to deny positively the possibility of such a combination in childhood, we call attention to the striking fact that latterly such cases are becoming even more rare. Some authorities deny that such cases occur—an absolute negative we do not consider justifiable; but it is sure that *in a large number of these cases miliary tubercles in the pia have been overlooked.* Now, when we consider that meningitis of the convexity without miliary tubercles complicates cheesy affections of the lungs—which may contain miliary tubercles too; when we remember that this very form of meningitis of the convexity involves only a very moderate purulent infiltration of the pia; when, on the other hand, we remember that only a very small number of the cases of tubercular meningitis without tubercles are genuine, it is highly probable *that the two classes of cases resolve themselves into one.*

8. *Dysentery.*—Doubt has been thrown of late on the existence of dysenteric meningitis. Firstly, the data are very few, and severe epidemics of dysentery have supplied no evidence; and secondly, it is possible that the existent data rest on incor-

rect observations (see the remark on meningitis in Heubner's Treatise on Dysentery).

9. *Diphtheritis.*—In the present state of our knowledge of diphtheria it cannot be positively decided whether purulent meningitis is a direct result of diphtheria or no. In the majority of cases the condition of the brain and cord after this disease resembles parenchymatous inflammation of the central organ: hemorrhages constitute the characteristic appearances, and are found in the membranes as well as in the cortex, in the whole brain substance, in the medullæ oblongata and spinalis, and in the intervertebral ganglia; disseminated myelitis of diphtheritis (Oertel), of variola (Lewinsohn, Westphal).

Occasionally meningitis seems to occur more indirectly, as a manifestation of pyæmic blood-poisoning. It is manifest that it is scarcely possible to express oneself positively in regard to every case resulting in pyæmic infection, so-called, as long as the present views of diphtheria are the prevailing ones. Politzer reports a case of purulent meningitis after diphtheritis conjunctivæ, which was first complicated by purulent inflammation of the joints, then with that of the pia. We ourselves have observed the following case:

27.—A cook, aged fifty-one, is brought into hospital late in the evening with diphtheritis faucium of moderate intensity, high fever, and small, rapid pulse. Organs healthy; no brain symptoms other than headache. Cauterization according to the method then customary. In the night delirium, restlessness, groaning, attempts to get out of bed; no convulsions, etc. Toward morning, sopor; by six o'clock profound coma, rigidity of neck, and distortion of the eyeballs. Two hours later, death. *Autopsy.*—Purulent meningitis of convexity and base of moderate intensity; ventricles normal. Nothing abnormal about the veins of the diploë or the sinus.

In such cases we cannot explain how the infection of the pia was brought about.

10. *Acute exanthemata*—

a. *Measles.*—Genuine meningitis of the convexity is a very rare complication of measles. The authorities of the largest experience mention single cases of tubercular meningitis which they have seen, but reports of purulent meningitis of the convexity are extremely rare, and the etiology is entirely different from that of the measles themselves. We have already described lepto-meningitis infantum as occurring in this connection. À priori reasoning does not aid us in understanding why this latter remarkable morbid process should not go on to formation of pus ; indeed, it seems that it does actually do so occasionally. The vague data of most authorities do not permit us to form a positive opinion. Proof of simple coincidence—as most publications give it—is no longer sufficient. We need evidence as to the manner in which infection of the pia was brought about. Hence, we are in uncertainty as to the intimate causes of this form of meningitis, in spite of the reports of Mettenheimer, Rilliet, Spiess, Löschner, Krug, and others.

b. *Scarlatina.*—Some of the cases of meningitis which occur in the course of scarlatina have doubtless only an indirect connection with the scarlatinal process. Our limited personal experience applies only to children with extensive suppuration in, and sloughing of the connective tissue of the neck, so that we must characterize this form of meningitis as "metastatic ;" but the experience of many others shows that meningitis of the convexity complicates scarlatina under other circumstances as well. It has been sometimes observed at the commencement of the disease, at the time of the intense fever which belongs to the stage of invasion, and then runs an extremely acute course, and is rapidly fatal. Lepto-meningitis, before mentioned in connection with measles, also occurs in this stage of scarlatina, and the connection between the two processes is quite as obscure. Finally, meningitis of the convexity has been known to complicate scarlatinal nephritis and great anasarca ; its connection also with the primary affection is unknown.

11. *Typhoid fever.*—The meningitis of the convexity which occurs sometimes during typhoid has nothing to do with the typhoid process. The great weight of evidence goes to show that meningitis is incidentally set up only where there are complications which involve infectious purulent foci, which again render further transmission of the infectious material possible. At all events, it is a rare occurrence (4 : 250, Hofmann ; see also Buhl and Griesinger). In a case of our own there was thrombosis of the crural vein with puriform softening ; chills followed, and then ensued purulent bronchitis, pleurisy, delirium, tremor of

the extremities, sopor, without slow pulse, and death. There were a large number of pyæmic abscesses in the lungs, no thrombosis of the pulmonary veins, meningitis of base and convexity of moderate intensity. In a case of Griesinger's—a girl, ten years old—there were pneumonia, about breaking down, and cheesy nodules in the bronchial and mesenteric glands; the meningitis was cerebro-spinal, and the ventricles were full of pus. Griesinger remarks that if caries of the petrous bone be present, meningitis may occur from this cause during the course of typhoid.

We have further known purulent basilar meningitis to befall a patient with typhoid, who had been most wretchedly cared for during his sickness, and whose upper jaw had become necrotic. Starting from the left superior maxilla, erysipelatous swelling of the left cheek and vicinity of the left eye set in; also great œdema of the lids, and on the second day *chemosis of the conjunctiva.* The inflammation made its way rapidly inwards through the orbit (unfortunately, it was not ascertained which of the structures contained within the orbit was especially affected), attacked the meninges at the base, and, with sopor, clonic spasms, and coma, caused death in twenty-four hours. At the autòpsy purulent basilar meningitis was found, and a large accumulation of pus on the left optic nerve, a small one on the right.

12. *Bright's disease.*—The liability of the various forms of nephritic inflammation to be complicated with inflammations of the serous membranes (pleuritis, peritonitis, endo- and pericarditis) is well known; meningitis, however, is a rarity. We have seen a typical case of this kind in a girl fourteen years old, who had acute fatty degeneration of the kidneys. Meningitis of the convexity, with trifling hydrocephalus, ensued on acute pleurisy, and its symptoms were few and obscure.

13. The scattered reports in medical literature of meningitis as a complication of whooping-cough (Bierbaum) lack anatomical proof, and are, on the whole, more or less doubtful. We do not deny that serous transudation occurs, but cannot consider that condition a true inflammatory process.

Pathological anatomy.—There is but little uniformity in the degree of development of the morbid changes. The amount of blood in the pia is most variable, and in estimating its variations the same principles hold good which we have already discussed in an appropriate place. The quantity of blood in the brain varies greatly also. Many patients are already very anæmic; but, on the other hand, the ventricular pressure being low, an important factor in compression of the vessels of the brain substance is wanting.

The inflammatory exudation in the pia may be of all possible degrees, from a collection of pus which is scarcely appreciable to the naked eye all the way to the deposition of large quantities of it in the tissue. The term "meningitis of the convexity" is in this case also an improper one, since very commonly the base participates in the inflammation, and in some cases even bears the brunt of the affection.

The effusion into the ventricles also varies greatly both in quantity and quality. In some cases the fluid is not increased at all, and the wall of the ventricles is perfectly normal; in other cases it is decidedly increased, is opaque with pus, or consists entirely of pus. No changes in the ependyma are known with certainty, although occasionally vague mention is made of acute ependymitis in the reports of autopsies. But little is said about the condition of the choroid plexuses; in all of our own cases the changes corresponded with the quality of the effusion. The changes in the brain itself, apart from gross complications, are involved in obscurity. It is highly probable that inflammatory affections of the cortex exist, but they have not yet been clearly demonstrated. Future thorough investigation, it is to be hoped, will give us satisfactory insight into the contemporaneous changes which take place in the body; but their recapitulation, under the existent state of knowledge, would be of very subordinate value, seeing that any distinction which we may make between cause and effect in these cases is based only on assumption. In each individual case we should strive to discover the path which the infection has taken to get from distant points to the pia—an attempt which hitherto, in the great majority of cases, either has been unsuccessful or has never been made.

Symptoms.

It is here, too, utterly impossible to draw even an approximative sketch which would include all cases; indeed, in this case it is more difficult than in most cases, for in no form of meningitis are the signs, the duration, and the causation of the disease so variable. Often terminal meningitis is found on autopsy, though no suspicion was entertained of it during life; so, also, with regard to many other changes. The reasons for these variations in the symptoms are:

1. The extent and the degree of the inflammatory disturbance are alike in scarcely any two cases: in one case, after the inflammation of the pia has lasted several days, pus has been formed in scarcely appreciable amount; in another, after several hours' duration, we find large quantities of pus. The reason for this variation must lie in the initial disease; but an attempt to discuss this point would lead us too far.

2. It is by no means in all cases that compression of an appreciable degree takes place. This accounts for decided variations in the course of the disease; for, if there be no effusion into the ventricles, sopor is less marked and more transitory than if the effusion be notable.

3. Often the inflammation is confined to the convexity, the base and the upper part of the cord remaining intact; thus many very valuable diagnostic points are lost.

4. The disease appears during the course of various *febrile disorders*, and hence a characteristic stage of invasion is wanting in the great majority of cases. Besides this, very often some sort of brain symptoms were already present, depending chiefly on the high fever, and no such essential change is brought about in these by the meningitis as enables us to detect a new complication with certainty. For instance, if febrile delirium were present, difficulty occurs at once; if uræmic convulsions, in connection with scarlatina, have appeared, meningitis is generally overlooked; if we have pyæmic infection, with chills, to deal with, the chills do not aid us to detect the meningitis.

5. In the majority of cases we are dealing with a central organ, the conditions of excitability of which have already

undergone decided modifications. Diminished reaction and excitability may account for the fact that we now and then are confronted by a well-marked meningitis, which was shadowed forth during life by such indications only as would induce a cautious diagnostician to doubt rather than decide. For these reasons, it seems more advisable to dwell upon the symptoms which occur in connection with the different forms of the affection, than to attempt a general sketch covering all cases.

1. *Pneumonia.*—The cases which were attributed to cerebrospinal meningitis by Immermann and Heller had no especially characteristic symptoms. The appearance of the disorder on the scene had no relation to any particular period in the course of the pneumonia, but dated from the third, fifth, seventh, eighth day, and even later. In only one case was it ushered in by vomiting, soon followed by coma. In one case, on the eighth day of the pneumonia, furious delirium, pain in and rigidity of the neck, and contracted pupils, came on. Rigidity of the neck was also observed in other cases. Respiration was not modified by the complication, and in the majority of cases vomiting and retraction of the abdomen were absent. Paralysis was not noted, except, in one case, strabismus. Headache was almost constant; but petechiæ, roseola, and herpes were always absent. In seven out of the nine cases there was hydrocephalus; enlargement of the spleen in four. The seat of the pneumonic process was not constant (whole right side, three cases; whole left, two; both lower lobes, two; left lower lobe, one; right upper lobe, one). We subjoin a few cases of meningitis of the convexity, with pneumonia, in which no connection with the cerebro-spinal form could be made out.

28. Male, aged twenty-one. Great increase in the fever at the end of the sixth day; delirium and loss of consciousness for the first time in his sickness; occasional complaints of severe headache; contracted and feebly responding pupils; no paralysis or convulsions; after three hours, profound coma; eyeballs rolled upward; no vomiting; no rigidity of neck; no retraction of abdomen; no slowness of pulse at any time during the whole course; high fever, without definite type; agonal hyperpyresis; marked venous congestion of retinæ; duration, eight hours; meningitis of base and convexity, of moderate degree; no effusion into ventricles; no miliary tubercles; agonal œdema of the lungs; purulent pneumonia.

29. Male, aged fifty-nine. Severe double pneumonia; for three days active delirium. At the beginning of the second half of the eighth day, after a period of quiet lasting several hours, again delirium; attempts to get up and run away; unconsciousness and wandering; manifest hallucinations of sight and hearing; pupils contracted and respond badly to light; vomiting; gradually, rigidity of the peripheral muscles; voluntary movements possible, but carried out tremulously, slowly, and unskilfully; restlessness continues till shortly before death; clonic spasms of upper extremities; no retraction of belly. About an hour before death patient is comatose; pupils still contracted and do not respond at all to light; never slowness, but frequency and smallness of pulse; death with hyperpyrexia, and after death still further rise in the temperature. Duration, twenty-nine hours; intense meningitis of the convexity, trifling at the base; no effusion into ventricles; profuse suppuration in the right fossa of Sylvius; changes generally more marked over right hemisphere.

30. Female, aged fifty-four. On the tenth day, the temperature being moderate, new chills and rise in temperature to 40° (104° F.); complains of headache, noises in head; is soon out of her head; wanders and is sensitive to light; soon afterwards slight rigidity of neck, and retraction of head into pillows; pupils contracted and respond badly; after about eight hours, divergent strabismus and paralysis of left abducens; lies quietly, while unconsciousness gradually increases to profound coma, with irregular and stertorous respiration; after ten hours, difference in the pupils, the right larger; slight ptosis on right side; no vomiting; no retraction of abdomen; slight clonic spasms of muscles of fingers and toes; no peripheral muscular rigidity; after twelve hours, death in coma; fever in the beginning high, but falls in the agony; no slowness of the pulse at any time; meningitis of base and convexity, in the former very marked; moderate effusion into ventricles; no miliary tubercles.

31. Double pneumonia of six and a half days' standing; severe case; steady high fever without any remission; small, frequent pulse. Male, aged fifty-two. Prune-juice expectoration; the patient then becomes mildly delirious, the condition of the pulse and temperature remaining the same; no headache; contracted pupils non-respondent to light later the right less contracted; conjugate spasmodic movements of the eyeballs, which finally are directed upward and to the right; complete loss of consciousness; stertor; no rigidity of neck; groans when neck is rotated; no vomiting; no slowness of pulse; temperature steadily high; death in nine hours. (Diagnosis, meningitis). Autopsy.—*Except the ordinary disturbances of circulation, nothing whatever abnormal is found in the nervous system, either macroscopically or microscopically.*

These examples, which could be indefinitely multiplied, are sufficient to show the difficulty of recognizing the complication with certainty. *There was not a single symptom which was common to all the cases which have come under our knowledge,* *or has not been present and equally marked in other conditions of the brain.*

In our cases it was always later than the sixth day that meningitis appeared on the scene; the latest case is that above reported, on the tenth day. There is no constant initial symptom; moderate chills, intense headache, rapidly developed and mild delirium, have been observed; we have never seen vomiting as an initial symptom. One of the sure indications—if, indeed, we have the right to speak of surety at all—seems to consist in a fresh, undue accession of fever. Still, one case was observed in which the temperature was in no way influenced by the meningitis. The only characteristic feature of the delirium is that it generally early gives place to sopor; sometimes it ceases suddenly, in which case it is generally coincident with collapse, and death follows quickly. Now and then furious delirium has been noted. A slight degree of rigidity of, and pain in the neck is always a valuable indication, but still is not always to be depended on, and can be present in cases in which post-mortem no trace of meningitis is to be found. The pupils are generally contracted at the first; secondary bilateral dilatation, and difficult reaction we have never observed, though we have observed unilateral dilatation. This, however, as shown by Case 4, may be the result of a disturbance in the innervation of the nerve-centres, the function of the brain being disordered. Spasmodic movements of the eyeballs are untrustworthy; *they occur independently of meningitis.* The really reliable basilar affections seem to constitute the surest indication, *i.e.*, complete paralysis of the abducens or the oculo-motorius; but these are very rare. Headache is present in all cases till sopor supervene. One case was characterized by vomiting late in its course; retraction of the belly did not occur, nor did paralysis of the extremities. This latter, even should it occur, must be regarded with caution, for I have twice seen complete and unquestionable hemiplegia occur just before death, and nothing more than œdema was found to account for it in the brain. Paralysis of the facial or hypoglossus we have never noted; of convulsive symptoms only trifling tremors in the extremities; nor have we ever noted changes in the skin, or other concomitants of cerebro-spinal meningitis. There is nothing characteristic about either the pulse or the temperature; in not a single case was the pulse slow. The fever was always high, and resembled the fever of meningitis only in this respect, viz., in one case the temperature fell below the normal point, and in several others hyperpyresis was noted during the agony. *Choked-disk and swelled papilla are among the most reliable indications.*

We have also known meningitis to complicate an already complicated pneumonia, in one case with hemorrhagic pleuritic effusion, in another with thin purulent pericarditis. In these cases, too, the symptoms were very variable and indecisive.

There are no absolutely reliable diagnostic signs, and difficulty will always be met with in distinguishing between terminal meningitis and functional disorder of the brain occurring during the agony. A fresh access of fever, which is more intense than the original one, which appears suddenly and is ushered in by a chill, must always arouse suspicion; suddenly arising, mild delirium, wandering and hallucinations, are very important signs. Contracted pupils are important only in so far as they accompany other symptoms, and the same holds good if they become gradually unequal. Sopor and coma are very important if they appear quite a long time before the terminal œdema of the lungs; spasm in the muscles of the eyeball is unreliable, much more so than paralysis of the abducens or oculo-motorius, or even than rigidity of the neck. One should not make the diagnosis of meningitis, during the course of pneumonia, from one or from a few signs; but if the signs, which we have indicated briefly above, are associated, the diagnosis is permissible.

A gradual and progressive affection of the brain will be recognized only with great difficulty in the course of *ulcerative endocarditis.* If one bears in mind the multiple hemorrhagic infarctions of the cortex, and the fact that they can be followed by irritability or loss of irritability of the brain, which is in no wise distinguishable from a like condition dependent on meningitis, it is needless to insist further on *the impossibility of making a positive diagnosis.* If the symptoms be those of a general affection of the brain, the possibility of meningitis of the convexity should be remembered. Moreover, these emboli also occur in the pia and give rise to hemorrhage there, so that their mechanical effects are equivalent to those of meningitis.

Brief mention has already been made of *meningitis as a complication of rheumatism;* this event is rare, and has nothing in common with the ordinary disturbances of the nervous system which are observed in connection with acute rheumatism. These latter are:

1. Mild and quiet delirium, with moderate fever; more common in persons of a nervous temperament; no macroscopic foundation.
2. The delirium of hard drinkers, up to genuine delirium tremens. No inflammation in or about the brain.
3. Hyperpyretic delirium; no macroscopic foundation.
4. Psychical disturbance, depression with hallucinations and illusions, or with exaltation (rare), or insanity dependent on the depression and the cause of the anxiety and illusions. No macroscopic foundation, except disturbance of the circulation.

One should be on one's guard, then, and not make the diagnosis of meningitis, unless the chain of symptoms be complete. One of those diseases, in the course of which purulent meningitis has really been known to occur, must be co-existent (endocarditis, pneumonia with pleurisy, pericarditis).

Cheesy affections of the lungs.—We have already expressed ourselves as to the significance of meningeal processes, without miliary tubercles, in connection with these affections. The diagnosis of an inflammatory affection with hydrocephalus is, of course, easily made when there is tubercular meningitis; but it may be utterly impossible to distinguish it with certainty from meningitis of the convexity. The symptoms themselves are the same, and as regards acuteness and arrangement of symptoms, tubercular meningitis varies so much that many cases of it coincide exactly with ill-defined cases of convexity meningitis. For in the latter case we have before us frequently only a peculiar modification in the symptoms before death, from dullness of the mental faculties all the way to complete sopor; lack of appreciation of time and place in their several relations; mild delirium; a gradual sinking into sopor and coma without complaint of headache, and without evident symptoms of irritation, whether motor or sensory; these are generally the prominent symptoms

Now and then a more alarming sign appears, as rolling of the eyeball, ptosis, clonic convulsions of the extremities, or dilatation of one pupil; gastric and intestinal symptoms are generally wanting; vomiting is rare at so late a stage; constipation is often replaced by diarrhœa, dependent on intestinal ulceration. Superadded are the symptoms of advanced phthisis, a high temperature and a correspondingly rapid pulse, which does not diminish in frequency, even under the influence of considerable effusion into the ventricles.

The fact that no considerable effusion takes place, even when the necessary conditions are present, may well depend in part on diminution in the amount of blood, caused by the phthisical malnutrition, and, in part, on the not inconsiderable atrophy of the brain which often takes place in phthisical patients. Under these circumstances, before the general intracerebral pressure reachès a certain point, a larger collection of fluid must take place than when the anatomical relations are normal.

The rare cases in which meningitis complicates *diphtheritis* seem to be always obscure. They run their course generally under cover of malignant blood infection, with high fever and general head symptoms—mild delirium, sopor, coma, trembling of the extremities, picking at the bed-clothes, etc.; as far as we know, the diagnosis has never been made with certainty.

It may be possible, occasionally, to diagnosticate meningitis in the course of measles or scarlatina, if a distinct series of symptoms of irritation, and their gradual interchange with those of depression, be observed. Even if we can diagnosticate meningitis, it will be but rarely that we can go farther and specify the form of meningitis.

The above described lepto-meningitis infantum will first occur to one; special indications (cheesy inflammatory products, etc.) will here and there suggest the tubercular form, but meningitis of the convexity will be very rarely recognized.

The symptoms of scarlatinal meningitis are so variable and deceptive that a clear portrayal of the affection cannot be attempted; for all sorts of nervous symptoms are described, from sudden collapse and rapidly ensuing death, through all possible forms of convulsions, even to eclamptic attacks and tetanus. Many cases are most obscure, even with the aid of an autopsy, and statements about meningitis, without autopsy, are absolutely valueless.

In rare cases, meningitis, as a complication of *typhoid fever*, betrays itself by definite symptoms. In two cases under our own observation it began with a slight chill, vomiting, and intense headache; then ensued delirium—at first passive, but later becoming active,—hallucinations, and attempts at flight. In one case there was rolling of the eyeballs, and later divergent strabismus; in the second, trembling of the extremities, and some retraction of the head; in neither did the pupils present symptoms of value other than defective reaction. The temperature was high to the end, the pulse always rapid. In both cases there was purulent meningitis of the convexity, with large ventricular effusion.

Prognosis.—All the forms of meningitis which come under this head have a very unfavorable prognosis, a fact which is not surprising, when we consider the foundation on which they rest (pneumonia, endocarditis, pyæmia, etc.). The favorable results which some authors obtain in the treatment of meningitis, especially in children, are very astonishing. *We have not seen a single case of recovery* where the diagnosis was strictly limited to cases which did not admit of the possibility of a doubt.

Such procedures as that of Bierbaum are calculated to place the question in any light but the true one. This author loses only ten out of forty cases, and of the first sixteen cases which are included in his article, although several were fatal, he has not a single autopsy to bring in evidence; nor in connection with etiology does he adduce a single corroborative anatomical examination. All his remarks on meningitis as a complication of the acute exanthemata, rheumatism, etc., are worthless.

As for *treatment*, we can only repeat what we have already said in another connection, but shall return to this point at the end of the next section.

VI. *Meningitis of the Convexity, due to external and unknown causes.*

Besides traumatic meningitis, a class of cases of meningitis of the convexity, whose origin is either unknown, or at best involved in vague hypotheses, awaits our consideration. This form does not befall a system already modified by disease, but, on the contrary, a system in perfect health; hence, it is the purest form of purulent inflammation of the membranes, and can be contrasted with the tubercular form, as no form which we have thus far discussed could.

The etiology is clear in a small fraction of these cases. We are led, by a series of very convincing personal observations, to agree with many writers who hold the view that exposure of the uncovered head to the scorching rays of the sun may give rise to purulent meningitis. Although there is no satisfactory physiological explanation of the fact, we lay much stress on cases of adults, who, after being exposed bareheaded during hard work in the open field to the midday sun, are immediately taken down with an acute affection of the brain, and the autopsy reveals purulent meningitis of the convexity. The question may be asked—and an answer is in the present state of knowledge impossible—why, when so many people are exposed to the injurious influence, so few suffer from it.

Now and then meningitis of the convexity occurs, and the closest scrutiny fails to bring the cause to light. Under such circumstances one is apt to suspect that one is dealing with a sporadic case of cerebro-spinal meningitis, but the symptoms do not agree with such an idea, and the whole process can only be regarded as an inflammation of the pia, depending on some local, but quite unknown, inflammatory irritation.

Further, taking cold, thorough wetting, sitting on damp ground, have all been assigned as causes of the affection under consideration. Were it any other than Hasse who mentions these causes also among others, one would be inclined to discredit them. We have had no experience of the kind. That severe mental application can give rise to purulent meningitis we cannot affirm, but there is no question about the predisposition of hard drinkers.

Primary meningitis of the convexity seems to us essentially a disease of adult life, and the liability greatest in youth and middle age. As contrasted with this, meningitis of the convexity in children occupies a peculiar position. It cannot be doubted that absolutely the disease is more frequent in this period of life; but if we subtract from the cases of this affection in children, taken collectively: *a.* Traumatic cases. *b.* Meningitis extending to the pia from the bones of the skull. *c.* The meningitis which complicates pneumonia in childhood, and the meningitis which we have characterized as "pyæmic" (true pyæmia, phlebitis of the umbilical vein, suppurating wounds of the bones, etc.). *d.* Meningitis complicating the acute exanthemata—a number of cases will still remain the etiology of which is entirely hypothetical, often sought for in the most opposite directions—a sure indication that we practically know nothing about it.

Doubtless insolation plays a part in children as well as in adults (Rilliet and Barthez); doubtless great irritation of an impetiginous or eczematous eruption on the scalp may give rise to meningitis, though probably this latter form belongs in that category of cases in which inflammation spreads by contiguity; the most reasonable explanation being, that septic material is carried by the veins from the suppurating surface to the interior of the skull. Further, there seems to be something about cholera infantum which may give rise to purulent meningitis; the affirmations of trustworthy writers (*e. g.*, Bednar) are too positive to be lightly disregarded.

Meningitis has also ensued on vaccinia, and in this case also it is undoubtedly "metastatic."

Over and above all this, there are cases of purulent meningitis of the convexity in children, which run their course before our eyes without any assignable cause. It is full as well to be somewhat sceptical about the large figures of authorities, even of those who were in the habit of making autopsies, for miliary tubercles have been overlooked thousands of times. It seems to be well ascertained that the disease is more common among those who are poorly situated, and whose food and care are insufficent.

Of adults, men are more subject to the disease than women. It is a remarkable fact, and directly at variance with a point in etiology given above, that apparently more cases occur during the cold than during the warm season. Finally, the history shows in many cases that the affected individuals were of a nervous temperament.

An epidemic grouping of cases has also been mentioned in connection with this form of the malady, but careful investigation seems to us to show that this is incorrect; for in one class of cases, when meningitis of the convexity was really present, it was manifestly the cerebro-spinal form, and in the other class the data are so vague and hang together so little, that genuine meningitis of the convexity is scarcely to be thought of.

On *autopsy* we find most typical pyo-fibrinous exudation into the pia, and there is no variation of moment in the localization of the affection. For instance, we do not find that the base alone is involved; but, if there be a difference, it is rather on the side of the greater involvement of the convexity, where the exudation is often present in large amount. The amount of blood in the vessels varies greatly, and a more minute description would merely lead to a repetition of what we have already said.

Be it only remarked that the amount of blood in the vessels, bears a certain proportion to the amount of effusion into the ventricles in *about half the cases.* The greater the latter the more marked is *the secondary capillary anœmia of the pia and cortex*, a condition which has much influence on the symptoms during life. The character of the effusion is variable; it is not uncommonly purulent, though even then it can scarcely be said to consist of pure pus, but rather of a mixture of pus and serous intraventricular fluid in varying proportions. The cerebellar pia is involved in varying degree, and often the inflammation extends somewhat over the medulla oblongata, so that the anatomical appearances of cerebro-spinal meningitis are very closely imitated. In some cases the choroid plexuses are involved in the inflammation, pus being visible in their tissue even to the naked eye.

The pia can be separated only with difficulty from the surface of the cortex, small or large bits of which are torn off with it and remain hanging on the vessels, leaving loss of substance behind. Small capillary extravasations are especially noticeable, but beyond this the cortex, in the great majority of cases, exhibits no gross changes, especially when capillary anæmia has been brought about by strong intraventricular pressure. Microscopic changes are always present in the cortex, and in some cases are still more marked than in tubercular meningitis. We have noted modifications in the protoplasm of the ganglion cells, limited to the deposition of relatively large granular elements, and loss of shape; we have also noted a loss of cohesiveness and fluidity in the protoplasm, such as is not found in several forms of rapid encephalitis, for the simple reason that the duration of the disease is too short. In some cases the suppuration was very marked, so that a diffuse, yellowish-gray maculation was visible to the naked eye on section of the cortex—evidence of a most intense suppurative process.

There is also a high degree of emigration of the formed elements of the blood throughout all the layers of the cortex, as well as a diminution in the calibre of the small vessels, due to the generally increased pressure. The result is that the vessels are nearly empty, although the presence of large numbers of the cellular elements of the blood in the vicinity proves that intense hyperæmia was antecedent. This is particularly the case when the amount of the intraventricular effusion is great. Although symptomatic compression of the brain from this cause often does not occur, yet the patient finally sinks into unconsciousness. In this case no great effusion is to be expected. The diffuse inflammatory affection of the cortex is alone sufficient to annihilate its functional activity. What part is played by the above-mentioned changes in the ganglion cells we will not venture to discuss.

Emigration also takes place in the white substance of the hemispheres.

If the exudation be very great on the convexity, and if the plexuses be very little, or not all involved, occasionally the ventricular cavity is found compressed and diminished in size, it is said. We have never had an opportunity of verifying either this or distinct inflammation of the ependyma of the ventricles. Hydrocephalic softening is found, but in far less degree than in tubercular meningitis.

The other appearances in the organism throw no light on the origin of the meningitis. Once we noted commencing pneumonia. Cloudy swelling of the internal organs is often present in high degree, and the parenchymatous affection of the kidneys has a certain influence on the symptoms during life, since albumen is very often present in the urine. These changes are doubtless the result of the very high fever, as in various other processes.

Symptomatology.

We will begin with the report of some cases:

26.—Strong sailor, aged twenty-three: hard drinker, though in last twenty-four hours has drunk scarcely two pints of wine. The day before he fell ill—one of the hottest days in July—went down the lake with a load of stone, and worked a heavy oar bare-headed. During the day, till about 3 P.M., says that he felt perfectly well (never was sick before; parents alive and well; no neurotic tendencies in the family; no scrofula). At 3 P.M. moderate headache came on, and increased from one quarter of an hour to another, so much so that already after the lapse of an hour, work, and even locomotion, became impossible. The headache was general, and periodically increased to such intensity that he yelled and cried out that they should throw him into the water. Head somewhat red, and hot to the touch. Extreme weakness of the extremities ensued immediately on the headache, so that the patient could scarcely stand; also giddiness, and a sensation as if the ground were giving way under his feet, and as if he were being raised backward high up in the air. Vomited once on the boat, and was brought ashore to his brother. After he was put to bed, *a chill of about an hour's duration*, during which was unconscious, and after which vomited again. Toward evening attacks of intense headache returned, so that he cried and screamed; otherwise condition the same; severe dizziness; face flushed; conjunctivæ glistening; epistaxis; the latter is followed by some relief, but stops very soon.

8 P.M.—A large, powerful man, now lying quietly in bed; decubitus dorsal; appears to be somewhat unconscious, but can easily be induced to give his attention and answer questions; sensorium normal. Complains chiefly of insupportable pain in his head, and dizziness, paroxysmal in character, and lasting about a quarter of an hour, and closing with nausea; pain, however, does not disappear entirely. Groans, and screams with pain during the paroxysms. Face much injected; head hot; temporal and carotid arteries strongly pulsating; eyes glistening and injected. Pupils are alike, and react well, though both are rather contracted; the muscles of

the eye, as well as those supplied by the facial and hypoglossus, normal; tongue with thick white coat. No convulsive movements of the extremities. The vomitus consisted of merely the contents of the stomach and some undigested particles of food. Heart, lungs, and abdomen normal. Temperature 39° (102.2° F.). Pulse 92, full and non-compressible. No albumen in urine. Eight leeches to head; ice bladder; large doses of compound infusion of senna. Application of leeches was followed by some relief.

At 10 P.M. sudden appearance of general convulsions. Patient is unconscious; his head is drawn backward into the pillows; neck is rigid; pupils of medium size, and non-respondent; the balls rolled upward; no strabismus; color of face is unchanged; no spasm of muscles of respiration. In the muscles of the eye, tonic spasm (the eyeballs rolled upward); clonic spasms of the muscles supplied by the facial and hypoglossal nerves; tonic spasm of the muscles of the neck and back, giving rise to moderate opisthotonos; finally, clonic spasms of great severity in all four extremities. This condition lasted about twenty minutes, and unconsciousness continued after the attack. Temperature 41° (105.9° F.). Pulse 104; small. A change of color in the face; patient inclined to cyanosis; nose and lips livid; respiration slow and stertorous. Although no râles were to be heard over the lungs, he was bled to the extent of twelve ounces, and then, as he had not roused from sopor, a tepid half-bath was given him in a large wash-tub, and cold water was energetically showered over his back and shoulders. After this consciousness partially returned; he again performed voluntary movements, and answered questions with partial correctness. Ice bladder; infusion of senna. Again in the night the convulsions returned, and the showering was repeated by the attendant, with the same success as before.

Second day.—Lies doubled up in bed; is not unconscious, but wanders, and has no recollection of what passed in the night; moves his head very slowly and carefully, on account of the severe pain in his head, which still persists. Reasoning processes are carried on slowly and clumsily, and often cease abruptly at the simplest answer; he needs time for consideration and often the power of reflection manifestly leaves him altogether. Knows where he is; cannot estimate the duration of his illness, but is remarkably clear as to all which preceded it. Frequent attacks of vertigo, and bilious vomiting twice during the examination. Now no rigidity of neck, but pain along the cervical vertebræ, spontaneous as well as evoked by pressure. General hyperæsthesia; cannot bear to be touched; pain in all his limbs, and even on the surface of the skin, especially severe over belly and legs. Skin hot and dry; tâches cérébrales.

Pupils of medium size; equal; respond slowly. Nothing abnormal about the muscles of the eye, or those supplied by the facial and hypoglossal. Has perfect power of motion in all extremities; complains only of subjective weakness; gait is tottering from weakness of the knees and vertigo (a sensation as if the ground were vanishing from under his feet); the power of pressure in his hands is very feeble, although his muscles are large and firm; spleen small; no eruption on skin; copious thin alvine discharges. No albumen in urine. Temperature 39.2° (102.8° F.).

Pulse 92, full and tense. All internal organs normal. Ice bladder; infusion of senna.

Evening.—A quiet day; no convulsions; several times an increase in the pain in head, causing him to groan and cry out. Vomited once; great thirst. Condition of sensorium unchanged. Respiration normal; no stertor. Thin dejections. Temperature 39.6° (103.3° F.). Pulse 104, full. A small dose of chloral.

Third day.—Delirious in the night. While lying still he talked continually about all sorts of things connected with his occupation, and once tried to get out of bed. Is still unconscious and wandering; when let alone talks disconnectedly about all sorts of things. Face rather pale; skin hot; inequality of pupils, the right being larger and responding more feebly than the left; no strabismus; unimpaired power of moving the eyeballs in all directions. Nothing abnormal about facial or hypoglossal. Trifling rigidity of neck, which is easily overcome, though not without causing pain. About every fifteen minutes loud complaint of severe pain in head; vomited once. Weakness of all extremities; when put on his legs, totters and sways backward; can scarcely hold himself erect for an instant. Tongue thickly coated; internal organs normal; two thin dejections, the patient asking for the bed-pan. Bladder full, emptied by catheter; urine cloudy and concentrated; albumen in considerable quantity; sediment consists of urates only. No enlargement of spleen; no eruption on skin. Temperature 39.4° (103° F.). Pulse 82, full and regular.

Retina.—Marked congestion; contour of disk ill-defined, especially the right; veins much dilated; arteries contracted. No traces of neuro-retinitis.

By exclusion of other brain affections, from the want of any other exciting cause than insolation, and from the interagreement of all the symptoms, the diagnosis of simple purulent meningitis is made.

Evening.—Temperature 40° (104° F.). Pulse 86, full and regular. Unconscious and delirious the whole day; nothing abnormal about the nerves of the face, but general rigidity of the muscles of all the extremities in slight degree, and moderate rigidity of neck; no convulsions.

Fourth day.—After quiet and mild delirium, toward morning respiration became irregular and deeply stertorous. *Is in a condition of sopor;* can be aroused for a moment with difficulty, but, groaning, relapses immediately into unconsciousness. Face pale; conjunctivæ injected and pouring out secretion; no chemosis. Right pupil dilated and non-respondent, though not completely so; left pupil responds feebly and is of medium size; condition of retina the same as yesterday; congestion and swelling of papilla marked; no signs of neuro-retinitis. Tongue with thick white coat, protruded in median line. Slight distortion of left angle of mouth. Feebleness of extremities; patient is put on his feet, but falls immediately in a heap; afterward moderate rigidity of extremities; slight rigidity of muscles of neck and back. Tenderness, but no retraction of abdominal muscles; no impairment of contraction in diaphragm, but irregularity, as in the respiration generally. Lungs, heart, and liver normal; no enlargement of spleen; no cutaneous eruption. Urine is drawn with catheter; albumen; under microscope vesical, but no renal, elements. Fæces passed in bed. Vomiting has ceased. Mobility of extremities alike on both sides. Temperature 39.6° (103.3° F.). Pulse 72, full and regular. At noon a moderate convulsion, beginning in course of the left paretic facial by clonic spasm of several minutes' duration; then clonic spasm in left arm and leg; then in right leg and arm, the right half of the face remaining free. This lasted about five minutes, and was accompanied by rolling of the eyeballs and complete unconsciousness; at the close sopor became deeper.

Evening.—Cannot be aroused from sopor; groans frequently. Other nervous symptoms are unchanged. Temperature 41° (105.9° F.). Pulse 74, full and regular. A cold shower-bath had no effect on the sensorium. Temperature 40° (104° F.), after the bath.

Fifth day.—Was quiet all night, with snoring and regular breathing. Temperature 39.8° (103.8° F.). Pulse 102, full and somewhat irregular. Patient is comatose; rigidity of neck is more marked; no longer groans when head is rotated. Right pupil dilated and non-respondent; eyeballs turned upward; no strabismus. Paralysis of right facial more marked.

Some of the groups of muscles of the right side of the body are tense; the arm can be abducted only with difficulty, and flexion is impaired in arms, as well as in hip and knee. Left side free. Cannot be aroused. Much albumen, but no renal elements in urine; fæces passed in bed; no return of vomiting. Spleen not enlarged; no cutaneous eruption, but a threatening bed-sore. Heart and lungs normal. Respiration irregular and stertorous. Nothing new about the eyes; no chemosis.

M.—Temperature 40° (104° F.).

P. M.—Temperature 40.2° (104.4° F.). Pulse 112, small and irregular. Nothing new except increasing pallor; no more convulsions; the muscles of the right side still rigid.

10 *P. M.*—Temperature 41° (105.9° F.). Pulse 132, thready.

12 *P. M.*—Cessation of respiration, which had become intermittent.

Autopsy.—Diploë moderately hyperæmic. Calvarium thick; dura perfectly normal; longitudinal sinus empty.

A cobwebby exudation is spread like a vail over the surface of the arachnoid; a moderately thick yellowish-gray fluid can be scraped off with the knife, and is found to consist of pus cells with a trifling amount of liquefied intercellular substance. The pia of both convexity and base much infiltrated with pus, especially along the course of the vessels. The pia soft, friable, and adherent to the cortex. Meningeal extravasations up to one centimetre in diameter on some portions of the convexity. The accumulation of pus is, on the base, more marked on the right side, and extends into the right fissure of Sylvius. Posteriorly suppuration extends on the medulla oblongata as far as the middle roots of the spinal accessory nerves. The cerebellar pia is less affected, except in the vicinity of the corpora quadrigemina, where the disease is very marked. The plexuses are very hyperæmic, dotted with some spots of suppuration; in the ventricles a moderate quantity of opaque fluid containing pus cells; the ventricles moderately distended, especially the posterior horns. A moderate number of capillary hemorrhages in the convexity of the cortex, which seems to be somewhat swollen and hyperæmic; beyond this no macroscopic change was detected, and a microscopical examination was not made.

Thoracic organs normal, except œdema of the lungs and a few subpleural ecchymoses on the posterior border.

Liver and kidneys healthy, but subsequent microscopic examination showed moderate fatty change in the latter. Spleen not hypertrophied.

27.—Student, aged sixteen. He was foolish enough, on a Sunday, one of the hottest days in July, to paddle round in the lake from 1 to 4 P.M., lying on his belly on a board. At 5 P.M. began to feel badly. Shoulders, back, neck, and upper arms, those portions of the body which were not protected by the water, severely sunburnt; the pain which resulted from this rapidly increased till it became insupportable. Violent headache, especially in the occipital region, came on at 6 P.M.; head red and hot to the touch; glistening eyes. About 7 P.M. a chill, lasting half an hour. A very restless night; suffered greatly from intense pain in head and dizziness; vomited once toward morning. The cutaneous pain continues as severe as ever.

First day.—A robust young fellow; upper arm, neck, face and back dark red; the skin swollen, very painful and tender. Injection of face and conjunctivæ; pupils contracted and respond; no redness of scalp. Very intense headache and the whole scalp tender on pressure. Muscles of eye as well as those supplied by the facial and hypoglossal nerves, normal. Vomited again this A.M.; tongue thickly coated; nausea. Internal organs normal. No further nervous symptoms. Temperature 38.5° (101.3° F.). Pulse 96, full and hard. Leeches to head; ice bladder; carron oil applied to sunburnt parts. Cutaneous pain rather less; headache persists; dizziness; nausea; has vomited once; pupils equal; he is somewhat sleepy and irritable; answers laconically; seems to forget everything.

Second day.—Mildly delirious during the night; sleep was broken. The erythema from the burn has faded somewhat. Motion of the neck causes pain, and the muscles of the part are apparently somewhat rigid. Headache very violent; cries out and groans. Vomited once. Pupils equally contracted, respond more feebly; muscles of eyes, facial and hypoglossal intact. Great injection of face and conjunctivæ. Hyperæsthesia of the entire cutaneous surface, as well as of the muscles and bones; any kind of motion is very painful. Is somewhat unconscious and wandering; appears not to understand some questions; answers shortly, and sometimes incorrectly. Internal organs normal; spleen not enlarged; no cutaneous eruption. A small amount of albumen in urine. Temperature 38.8° (102° F.). Pulse 96, full.

Evening.—Was delirious all day, but lay quietly in bed and vomited twice. Pupils equal, contracted; respond badly. No new motor or sensory symptoms. Temperature 39.8° (103.7° F.). Pulse 92, full.

Third day.—Very delirious all night; sang, yelled, and screamed; is now wandering; lies in sopor, and mutters; eyes closed. Face paler and cool; conjunctivæ hyperæmic. Pupils equal, less contracted, respond very feebly; no strabismus or ptosis; facial and hypoglossal normal; no more vomiting; rigidity of neck. Moves his limbs himself, but passive motion causes pain. Tâches cérébrales on belly and legs on slight pressure. The erythema caused by the sun is less intense; the epidermis is peeling off the neck, leaving excoriations. Groans often; respiration is slow and snoring. No enlargement of spleen; passes his water in bed; a specimen removed by the catheter contained a trace of albumen. Temperature 39.4° (103° F.). Pulse 86, full, rather irregular.

Evening.—Condition unchanged. *The right pupil the larger of the two, and completely non-respondent.* No strabismus or other motor morbid phenomenon. Temperature 39° (102.2° F.). Pulse 82, full, and rather irregular.

Fourth day.—Sopor; can scarcely be aroused; every irritation or every motion calls forth groans and cries. Rigidity of neck; pupils as yesterday. Retina: moderate congestion of both retinæ; no hemorrhage; no swelling of the papillæ; no signs of neuro-retinitis. No strabismus or other motor or sensory symptoms; power of swallowing by reflex action intact. Facial and hypoglossal normal; rigidity of neck has disappeared. Temperature 37.8° (100° F.). Pulse 86, full and regular.

Evening.—Condition unchanged. Temperature 37.6° (99.8° F.). Pulse 88, full and regular.

Fifth day.—A quiet night without delirium, sopor less marked. He can again be partially aroused; turns his eyes toward a visitor; his expression is extremely dull; he does not reply to questions. No rigidity of neck. Right pupil is still the larger, but now responds somewhat, though not as well as the left. Facial and hypoglossal normal. Hyperæsthesia is diminished. Urine and fæces passed in bed. Nothing like a convulsion. Temperature 37.5° (99.6° F.). Pulse 82, full, and regular.

Evening.—Temperature 37.5° (99.6° F.). Pulse 82. Consciousness gradually returning; sopor diminished; replies shortly to questions; has again vomited once.

Sixth day.—A restless night, not delirious, but sleepless. Sopor has nearly disappeared. He can tell his name, etc., though he is still wandering and stupid. Pupils of medium size, equal, and respond better. Passes his water in the vessel, and can stand. Complains of pain in head. Temperature 37.2° (99° F.). Pulse 76.

Seventh day.—Completely conscious. Memory returns, remembers the cause of his illness, but does not know how long he has been ill. Less headache. *Pupils normal.* Has some appetite.

Improved daily, and after a week had recovered entirely.

We will not attempt at present a connected account of the symptoms, deeming it better to postpone this till we compare the symptoms of meningitis of the convexity with those of tubercular meningitis. The only absolutely reliable sign is the presence of tubercles in the choroid; but this, as we have seen before, is very rare, since for some reason or other tubercles are found in the choroid in very few cases of tubercular meningitis.

I. *Age and frequency.*—Simple purulent meningitis of the convexity is by no means a common disease either in adult or early life. If we subtract all those cases which are secondary to other affections, very few cases remain in which the disease is primary. The view, then, of Bierbaum, that the disease is most common in children under two years of age, must be essentially modified. He considers the disease less common in the second period of childhood, and more frequent again in youth and manhood. With the latter of these three propositions we must agree, but if the liability to the primary form is as limited as he says to the first period of childhood, this form of meningitis of the convexity is, in general, very rare, while the liability increases decidedly from puberty on. In this respect tubercular meningitis is very different. Apart from the far greater absolute frequency of this latter affection, the liability to it is greatest between the second and seventh years; and hence, if absolute frequency is to decide the question, the greater probability will always be in favor of tubercular meningitis. Moreover, when discussing whether we have meningitis of the convexity to deal with, let us always bear the etiology in mind; and, when possible, distinguish between the primary and secondary forms, which, as we said before, it will be possible to do in only a fraction of the cases which come under our notice.

II. *Antecedents.*—Meningitis of the convexity occurs as an acute affection in robust persons who are in full health; in some few cases the etiology assists us in making a diagnosis, though in Case 26, reported above, the diagnosis would certainly have been made even without the previous exposure to the sun. In other cases the etiology is involved in complete obscurity, and the secondary forms have their own special etiology. The fact that those who are the subjects of the primary form are generally strong and healthy people, may to a certain degree aid us in making a diagnosis. A large number of the subjects of tubercular meningitis bear plain evidences of old scrofula, or of recent tuberculosis. Glandular cicatrices and swellings, affections of the bones, all sorts of chronic inflammation with cheesy products, are full of meaning; but also full of meaning is the fact that persons fall victims to this disease who are in the flower of their youth, and free from evidences of constitutional taint; many such cases are on record. Hence it is only with great caution that we can avail ourselves of these points for purposes of diagnosis.

III. *Prodromata.*—Primary meningitis of the convexity has few prodromata. Its commencement is extremely acute, and often marked by a chill. If it be secondary, tubercle, at any rate, is excluded by the antecedents. This acute commencement is of fundamental importance in contrasting the disease with tubercular meningitis, the commencement of which is in the great majority of cases very different. In persons of whatever age, general and slowly progressing disturbance of nutrition is present: they lose strength, look badly, become gradually anæmic, diminish in bodily and mental vigor; or very often evidences are present of gradually beginning and progressing tuberculosis, such as glandular or pulmonary affections, suspicious chronic bronchitis attended by fever, hæmoptysis, disease of the bones, etc. Still, it is a matter of experience that all the above signs may be wanting, and the disease rapidly reach an advanced stage. This guide too, then, is not unfailing for diagnostic purposes.

IV. *Initial symptoms.*—Primary meningitis of the convexity begins its course very acutely, with a rapid rise in temperature, and often chills. Intense pain in the head comes on almost immediately, and, after the lapse of a few hours, the patient may be unconscious and delirious. Secondary meningitis, too, often causes a very sudden change in the symptoms of the primary affection, though sometimes such is not the case (see above), and a diagnosis is impossible. The tubercular form begins much less acutely. The striking change in disposition of children which often precedes—and that such a change does occur we consider beyond the possibility of a doubt—is well known, and has already been alluded to. In adults, also, an insidious approach is much more common: a chill is a rare exception; a gradual rise in the temperature the rule; the first symptoms, indeed, are not alarming.

V. *Duration.*—This is very short in primary meningitis of the convexity, which seldom lasts more than eight days. In the secondary forms death may ensue in from twelve to thirty-six hours, while tubercular meningitis generally lasts from eight to fourteen days. Variations from these figures upward or downward have been already touched upon. The difference in duration may be, then, but trifling, and must be regarded with caution.

VI. *The course of the temperature.*—Wunderlich and Rosenstein have laid down rules as to the temperature in meningitis of the convexity, which are universally accepted. The fever is in direct ratio to the extent of the process (Rosenstein); if the latter be intense and involve the whole pia, the temperature generally is as high as 40° (104° F.) on the third day, and remains at this point with slight variations. A further rise at the approach of death is inconstant (Rosenstein); so likewise a further rise *post-mortem.* The course of the temperature in the tubercular form is much more variable; at the commencement it rises gradually, and this we would consider precisely the normal course in cases which are not complicated by a febrile affection of earlier origin. The absolute height which is attained is moderate. Marked morning remissions, inexplicable variations upward or downward, even remissions to the normal standard for days at a time, are met with. The temperature rises during the agony, and falls during collapse; in the latter case it often rises after death. The action of antipyretic remedies is considered by Rosenstein the best gauge of the intensity of the fever. Sometimes their action is slight; sometimes zero. Even when they do act, they do not postpone the fatal result, which takes place all the same, only that the temperature is lower.

VII. *Pulse.*—Provided there be no compression of moment, the pulse corresponds pretty closely to the temperature; but from the very commencement a slow pulse, very little influenced by the rise and fall of the temperature, has been noted. If considerable compression take place, the effect on the pulse is about the same in the two diseases under consideration, and the pulse is not available in differential diagnosis. Ventricular effusion may take place in the course of either affection, though it is more common in the tubercular form, and it is on this effusion that slowness of the pulse depends. In secondary forms of meningitis of the convexity a slow pulse is very unusual. The pulse rises and becomes irregular toward the termination of both affections, and hence during the final collapse there is much divergence between the course of the pulse and that of the temperature.

VIII. *Mental symptoms.*—It is a common idea that these are more violent in simple than in tubercular meningitis. It is, on the whole, true that the delirium and those symptoms which are dependent on reflex action are more violent in meningitis of the convexity (attempts at flight, resistance to interference, intemperate language); also that, in correspondence with the whole course of the disease, they give place more rapidly to complete loss of consciousness. The large number of cases of tubercular meningitis which are characterized by precisely the same symptoms shows how very unreliable these symptoms are. The greater the experience of the observer, the more cautious he is. The duration of the violent delirium, too, varies so much in both affections, that it is of no diagnostic value. The most that we can say, then, is that the delirium becomes more rapidly maniacal in meningitis of the convexity than in the other form; that in the majority of tubercular cases the mental disturbance increases slowly; and—the most important point of all—that subsequently, and as a result of treatment, remissions occur.

IX. *Retina.*—In rare cases the diagnosis is established beyond a doubt by the discovery of tubercles in the choroid. The other changes vary in rapidity of development, reaching their height more quickly in meningitis of the convexity. As to whether neuro-retinitis is more common in one affection than in the other, we will not venture to express an opinion.

X. *Motor symptoms. Paralysis.*—In meningitis of the convexity paralysis is far from common, and facial paralysis (see Case 26) is far more rare than in tubercular meningitis, in which affection this symptom, as we have shown already, is very often present. The localization at the base accounts for this fact, and, although we do sometimes find facial paralysis in meningitis of the convexity, as a rule the case terminates fatally too soon for the function of the nerves which spring from the base to be completely lost. Inequality in the innervation of the pupils is often observed in meningitis of the convexity; but we have never seen either one or both pupils dilated *ad maximum.* The function of the facial nerve is often slightly impaired, and sometimes again restored; besides this, we have seen something analogous as regards ptosis. Paralysis of the extremities, hemiplegia, weakness of an arm or leg, have all been noted, but have never come under our personal observation.

All these conditions are more common and highly developed in tubercular meningitis. The facial paralysis, we repeat, is not dependent exclusively on the localization at the base, and paralysis of the extremities is dependent generally on special circumstances, which are in close connection with the fundamental disorder.

Convulsions.—The part which these play in meningitis of the convexity is an important one. Experience has forced us to this opinion, although it differs from that of other authorities. Nobody hesitates to accept the applicability of this fact to early life, and it is especially during the first and second years that convulsions are common. Rilliet and Barthez call particular attention to the frequency of convulsions in this period of life, and claim for these forms a very rapid course (from two to five days)—the convulsive or eclamptic form of Bierbaum. Another form, which occurs chiefly between the fifth and tenth years, is less frequently characterized by convulsions, has a more protracted course, and is more commonly accompanied by delirium (the phrenitic-comatose form of Bierbaum). Still, convulsions sometimes occur in this form too, and how much value there is in such a classification is easy to see. The intensity and distribution of the convulsions are susceptible of a very considerable variation, all the way from slight clonic spasms of the extremities, grinding of the teeth, and slight twitching of the angle of the mouth, to eclampsia of long duration, with complete loss of consciousness—if, indeed, the patient were still conscious—with epileptiform twitchings of all the muscles of the face and extremities, often with cessation of the respiration (tetanus of inspiration and expiration), with clonic spasm of the respiratory muscles. Such convulsions as these certainly spring from the medulla oblongata, while partial twitchings in particular groups of muscles spring rather from the cortex.

Convulsions form as prominent a symptom in tubercular meningitis as in meningitis of the convexity in early life. The form of the convulsions is precisely the same, and the only difference is that they are somewhat less frequent in connection with the tubercular form.

In adults the condition of affairs is different. Our experience shows that convulsions are more common in meningitis of the convexity (as in Case 26) than in tubercular meningitis, in which latter affection they are the exception. In connection with other symptoms, this fact may be of decided value for diagnostic purposes. We are fully conscious that other authorities (for instance, Rosenstein) do not agree with us in this opinion, which we base on the analysis of a large number of cases.

Contractures.—Rigidity of the neck is a usual symptom of both affections, but comes on sooner, and is more marked in meningitis of the convexity. Retraction of the abdominal muscles is a rare exception in simple, and is seldom absent in tubercular meningitis.

General rigidity of the muscular system is common in simple, rare in tubercular meningitis. Partial contractures seem to occur indifferently in either affection.

XI. *Sensory symptoms.*—On this head, in the nature of things, there is but little to be said in connection with either disease. Hyperæsthesia of the skin and periosteum is very common in simple, less so in tubercular meningitis; but our knowledge is so limited as to partial anomalies of sensations, that they offer no point of distinction between the two affections.

Headache and vertigo are about equally marked, although the former is rather more amenable to treatment in the tubercular form. Of course, as soon as sopor comes on, sensation can no longer be tested, and in like manner, in both affections, reflex action is lost since they are terminated indifferently by paralysis of the reflex centres of the medulla. Hallucinations are also equally common in the two affections.

XII. *Vomiting and constipation.*—These are valuable indications, point indifferently to either affection, and are rarely lacking in either.

XIII. *Respiration.*—Greatly increased frequency of respiration has been dwelt upon as a diagnostic point in meningitis of the convexity, especially in children (*jagende Respiration* of Bierbaum); we have not observed it in adults. As in the tubercular, so in the simple form, the Cheyne-Stokes type of respiration sometimes occurs, especially when sopor lasts for a considerable length of time. Irregular and sighing respiration occurs in both affections, and is characteristic of neither; the same holds good of the *cri hydrocéphalique.*

XIV. *Skin.—The tâches cérébrales* are equally common in both affections; cutaneous eruptions are rare in both, and have no diagnostic value. Herpes is rare in either, but seems to be rather more common in meningitis of the convexity.

XV. No decisive indication is to be obtained from *the spleen;* its size varies in both affections, but enlargement seems to be rather more common in the tubercular form.

XVI. We have no thorough examination of the urine in meningitis of the convexity at our command; we have had opportunity to demonstrate only the large amount of urea; several times we noted the presence of albumen, to which attention has been called by Rosenstein. Whether an increase in the phosphate of lime, which was several times noted by Oppolzer, is constant or no, we are not prepared to state.

Prognosis.

If we bear in mind the fact that there are a fair number of cases recorded in literature similar to Case 27 above reported, we are justified in holding the opinion that the prognosis in meningitis of the convexity is not absolutely unfavorable; and, indeed, there cannot be much doubt that in Case 27 we really had meningitis of the convexity to deal with, especially when we consider how closely the etiology of the affection in general agrees with that of Case 26. The possibility of the arrest of the process at a stage when actual destruction of tissue has not as yet taken place, and reabsorption can still come about in the pia, pathological anatomy offers no grounds for denying. We are thus led to the opinion that the prognosis is less unfavorable in this than in any other form of meningitis; but it is only in cases which had not reached their full development that recovery has been observed, and if the disease be fully developed we consider recovery simply impossible.

Treatment.

In accordance with what we have just said, we can hope for more from treatment in this form of meningitis than in all the others put together.

The means of treatment group themselves under three heads:

1. *Antiphlogistic.*—We have already mentioned several times the therapeutic measures which come under this head: leeches to the temples or the mastoid processes in sufficient number, ice-bags to the head, and shaving the head, are the chief measures. This is, moreover, perhaps the only form of meningitis in which *venesection* is indicated. If the subject of the disease is a robust man, if the invasion be very acute, if the symptoms be steadily progressive, if the mental symptoms be very marked from the commencement, if the fever be high and the pulse rapid and full, venesection may be productive of much good, which, though striking, is unfortunately only too often merely transitory; as is also the effect of local abstraction of blood from the head. The latter, due regard being had to the strength of the patient, may be repeated several times. The intestinal canal may be acted on to supplement the above measures; the discharges should be as copious as is possible without injury to the intestines.

2. *Antipyretic.*—If the fever be high we should attempt to reduce it, and to obtain this end repeated cold baths are the best means. Many systematic experiments have been made with this object in view, and the measure seems worthy of confidence in this disease as well as in others. It is sometimes useful to place the patient in a lukewarm bath and shower his head with cold water from a small height; we have found this of decided benefit when there are severe convulsions. General principles govern the temperature and duration of the bath, before which the strength of the patient should be carefully considered, and during which the pulse and respiration should be closely watched;

to such cases the bath should be administered by the physician himself. Drugs may be used to supplement the antipyretic action of the cold bath, if they can be swallowed; in one of our cases rectal injections of quinine were given with some success. Occasionally salicylic acid and its salts may be used. As to the effect of baths on the whole course of the disease, we refer again to the remarks of Rosenstein.

3. *Symptomatic.*—If there be great mental excitement, an attempt should be made to quiet it, and this result will be brought about quite as well by cold affusion as by narcotics. Still, cautious administration of the latter is often of great benefit; we have seen small injections of morphia or weak rectal injections of chloral hydrate, from fifteen to forty-five grains (1–3 grammes), of service. Strong enemata of chloral hydrate, from two to four scruples (3–5 grammes), have a powerful effect, and are to be administered with caution. We have several times been obliged to combat unremitting convulsions, and small subcutaneous injections of morphia and enemata of chloral hydrate have stood us in good stead.

If coma last a long time, there is danger lest inanition accelerate the fatal result, the patient being unable to swallow. This is an indication for nutrient enemata, or for the introduction of a tube into the œsophagus through the nose. If the pulse be small and irregular, stimulants are called for; and if the patient cannot swallow, they must be given in enema, or tincture of musk may be injected under the skin.

Mercury and iodine are held in high favor in some quarters, but have not been beneficial in our hands. If mercury be administered, the best method is that recommended by Ziemssen in epidemic cerebro-spinal meningitis.

VII.—*Traumatic Meningitis.*

Before entering on the consideration of traumatic meningitis, we must glance at several groups of symptoms which often precede this affection, and which have many points of resemblance to one another. The conditions which are known as concussion, compression and contusion of the brain have an intimate connection with subsequent meningitis, so much so that in what follows we must necessarily touch upon them briefly.

We can make the following approximative classification of the forms of traumatic meningitis, according to whether an immediate connection can be shown to exist between the trauma and the meningeal inflammation, or according to whether several intermediate steps intervene (osteophlebitis, etc.):

1. *Meningitis subsequent to concussion of the brain*, without any injury to the bone or the soft parts, and without contusion of the brain, or its sequel—abscess of the brain.

2. *Meningitis subsequent to injury to the soft parts alone of the skull.* In this case secondary inflammation of the bone, or thrombosis of the veins of the skull, which has undergone puriform softening and extended to the sinus of the dura mater, forms the connecting link.

3. *Meningitis subsequent to a perforating injury.* It is a result of early decomposition in the open wound, and is analogous to acute inflammation of the connective tissue in the vicinity of contusions in general. In this case there is no doubt that we have to do with a primary infection of the pia, and hence it is this form of meningitis which has especially been characterized as primary.

4. *Secondary meningitis subsequent to a perforating injury.* This form makes its appearance later, and probably depends on secondary ostitis, the result of the trauma. In this case, too, venous thrombosis, extending inward through the bone, plays a principal part, though doubtless the pia is infected in other ways as well.

5. *Secondary meningitis subsequent to a perforating injury complicated by extravasation of blood.* As a result of acute ostitis, decomposition takes place in the clot between the bone and the dura. The dura, which is separated from the bone, may have already become necrotic, before the decomposition in the clot, or may become so afterward, and from this point the infection of the pia starts. Or else necrosis does not take place, but suppuration on the external surface of the dura, quickly making its appearance on the inner surface, and from there extending to the pia.

6. *Finally, meningitis may not appear on the scene till necrosis be established in the bone.* Exuberant and healthy granulations are of great importance in this connection; the fewer and more defective they are, the greater is the danger lest, if free discharge of the pus be temporarily checked, portions of it get to the pia, and there set up fresh inflammation. As contrasted with secondary meningitis, this form might perhaps be called tertiary.

7. *Meningitis subsequent to abscess of the brain.* An abscess may break through on the surface of the brain and set up purulent meningitis; this form might also be said to be tertiary.

1. *Meningitis subsequent to simple concussion.*

(*Commotio cerebri.*[1])

A great modification has taken place of late in the views regarding concussion. The more the conviction gained ground that scattered lesions of the brain substance cannot account for the general character of the symptoms in this affection, the more certain it became that in pure concussion there is no appreciable lesion, and the more was one forced to the conclusion that it is an anomaly of circulation which we have to deal with, which anomaly must persist as long as its symptoms, and must be recoverable from.

The old view of Littré, that concussion is a collapse, a shaking up and disorganization of the brain, is a matter of history, and no longer tenable.

We cannot pass over the view held by many physicians of the present day, according to which a severe jar of the whole head mechanically affects the brain; and molecular dislocation of the elements of the brain is supposed to account for the remarkable association of symptoms, a more thorough knowledge of which is despaired of on account of inadequacy of means of investigating it. "A dull and heavy blow inflicted on the head tends first to drive before it in the same direction the part of the skull to which the blow is applied; simultaneously the impulse is propagated through the bone, by reason of its elasticity, to the underlying mass of the brain; tends to drive this also before it in the same direction, and thus causes it to impinge against the opposite wall of the skull, by which, in like manner, it is again immediately rejected in the opposite direction. Thus is brought about a series of vibratory movements, which permeate the brain as a whole, as well as its individual component parts, and the intensity and duration of which depend on the character of the blow, and of that part of the skull to which it was applied." (Bruns.) Directly opposed to this view are the experiments of Gama, Alquié, and Fischer, who, having constructed an apparatus in as strict compliance as possible with the physical conditions of the skull and brain, came to the conclusion that in concussion the brain, as a whole, is displaced and driven against the opposing wall of the skull; but that vibrations, which permeate the brain and present the phenomena of reflection and interference, can have nothing to do with it.

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[1] *Fischer.* Sammlung klinischer Vorträge.—*Bergmann* (Billroth and Pitha, Surgery), p. 206.—*Bruns*, p. 748.

Further, Fischer establishes the theory that, in an organ of such structure and consistency as the brain, such vibrations could scarcely be set up without causing solutions of continuity and hemorrhages—conditions which are found in by no means all cases of concussion. Still further, he calls attention to a fact which cannot be denied, viz., that dislocation, shaking up, and solution of continuity do not lead directly to coma, but that this condition always depends on some complication.

The theory, too, of Fano and Nélaton, which was based on the discovery of capillary apoplexies, has never enjoyed more than partial acceptation. These apoplexies were shown to be far from constant, and even the further development of the theory by Stromeyer did not succeed in bolstering it up. He advocated the view that the apoplexies take place during the stage of reaction, not at the time of the concussion, at which time there is a solution of continuity in the fibres and vessels of the brain; but, owing to feebleness of the circulation, neither extravasation nor coagulation takes place.

Of late the question has reached a more satisfactory solution. Starting from the long well-known fact that the normal function of the cortex is dependent on unimpeded afflux of oxygenated blood, it was hoped to trace the cause of the concussion to *general disturbance of the circulation in the brain.* A mechanical expression of the blood out of the vessels was naturally thought of, for the brain is doubtless momentarily compressed, and thus the amount of nutrient fluid diminished. But the symptoms of compression are persistent, while actual compression resulting from a fall or blow on the head must be of but short duration, since the very elasticity of the skull would cause it to resume its normal shape almost immediately, and thus refill the vessels. Further, as Fischer correctly remarks, the exciting cause of concussion is not always such as to compress the skull, but may be a very trifling injury.

There are two physiological facts which seem to throw some light on this subject. The first is the affirmation of Nothnagel (denied by Jolly, Rigel, and Frey) that strong irritation of peripheral sensory nerves causes contraction in the vessels of the brain. We have already discussed this question, and ranged ourselves on the side of Nothnagel. His view had all the more applicability to concussion, as it seemed reasonable to suppose that spasmodic contraction takes place in the arteries of the brain as a result of severe irritation of the skin and bone on the periphery. Opposed to his view is the fact that the attempt to induce coma by reflex irritation of the vaso-motor nerves was not attended with success; still it is true that the conditions in the two cases—concussion and the experiment of Nothnagel—are by no means identical. Again, reflex contraction of the arteries of the brain is transitory, but the symptoms of concussion continue sometimes for days, and neither experiment nor observation teaches us anything about vascular contraction which lasts for days. But, Fischer has very ingeniously sought to explain the manifestations of concussion by a second physiological fact, viz., the well-known results of the experiments of Goltz on vascular paralysis. If we suppose that reflex vascular paralysis is caused by the injury to the head, both the symptoms during life and the anatomical appearances in fatal cases become comprehensible.

The anatomical appearances are very fragmentary. Cases have been observed in which the symptoms were very marked during life, and yet absolutely nothing abnormal was detected in the brain after death. Rokitansky and Nélaton found small scattered capillary apoplexies, but even the latter confesses that they are not constant. The most trustworthy observers (Bruns, Fischer) have noted manifest circulatory disturbance—marked arterial anæmia, and equally marked venous hyperæmia. *This is the sole constant appearance.*

The symptoms which belong to this cerebral condition are relatively simple. The subject of severe concussion is completely comatose, but continues to breathe and his heart continues to act, but the function of the cortex is suspended; consciousness is completely lost, as well as the power of appreciating an irritation. Nevertheless, there is no paralysis; voluntary movements do not take place, but the innervation of the peripheral muscular system is not completely cut off. The pupils are dilated and react, sometimes feebly, sometimes not at all. The countenance and extremities are pale and anæmic. In severe cases the patient cannot swallow, and no result follows the irritation of even so sensitive a surface as that of the conjunctiva. There is vomiting; the pulse is slow (50, 40, 30): in severe cases very small, in moderate cases of medium fullness; the temperature is normal. Respiration is often very gentle and superficial, often irregular, and interrupted by sighing inspirations; urine and fæces are either passed involuntarily, or else there is retention of urine. These symptoms make their appearance simultaneously with the fall or blow, do not increase, but persist always the same in the subsequent course, and the appearance of new nervous symptoms is evidence of a complication. The duration of this condition is most variable: sometimes, after a few minutes, consciousness, perception, and the power of voluntary movements, return; sometimes only after hours, or even days; indeed, cases are on record in which this condition persisted for weeks, and was still recovered from.

In mild cases recovery is sometimes rapid, in severe cases it is always gradual; sometimes improvement is followed by relapse, and this may be repeated several times, until finally permanent improvement takes place; the pulse increases in frequency and fullness, the respiration returns gradually to the normal standard, the first sign of improvement being occasional deep inspirations at intervals; the circulation is restored in the face and extremities; voluntary and reflex movements. and finally, consciousness, return. The patient can swallow again; the pupils become more contracted and respond to light. The gradual awakening of the sensorium is noteworthy; the patient is often as in a dream, and cannot follow out a train of thought, which vanishes completely almost as soon as it is entered on, but gradually the function of the cortex is restored. After this a condition is very commonly developed which has been called, not inappropriately, irritation of the brain, and which seems to depend on the intensity of the coma; the afflux of blood to the brain is increased, the face becomes red and turgid, the eyes glisten, the patient is restless, tosses about in bed, his movements are rapid and violent; sometimes he is a little out of his head, and often complains of headache. These symptoms may reach such a pitch as to arouse grave anxiety lest meningitis be imminent.

Sometimes transitory diabetes accompanies the concussion, and may even last several days longer than the symptoms of the latter; this was noted by Fischer in six out of forty-three cases, the amount of sugar never being very great. In rare instances diabetes insipidus ensues on diabetes mellitus, or is present from the commencement. Fischer reports several very interesting cases of this nature, in one of which polyuria lasted three months.

According to Fischer, albumen is a more common constituent of the urine than sugar, but this observer has never found any formed constituents other than red blood-discs, and a brownish detritus, which he considers the remains of disorganized blood-discs.

If it be asked whether Fischer's theory covers all these symptoms, we must confess that, though it is the only theory which can explain the symptoms in general, some of its anatomical and physiological basis is still incomplete.

Fischer reasons as follows:

a. The hypothesis accounts for the abnormal distribution of the blood, inasmuch as, if loss of vascular contractility be superadded to slow and enfeebled action of the heart, arterial anæmia and venous hyperæmia must ensue. If the vessels be paralyzed, the integrity of the circulation can be maintained only by increase in the force of the heart's action; the force, however, is not increased, and hence the arterial flow must be diminished and the blood must accumulate in the veins.

b. Further, the hypothesis accords fully with the fact that the cortex is completely paralyzed, while the medulla oblongata still performs its function, though imperfectly. Many facts go to show that the hemispheres bear the brunt of a shutting off of the arterial supply, while the medulla suffers much less, and is even stimulated.

c. He accounts for the absence of convulsions in concussion by the experiments of Kussmaul, in this way: that in concussion anæmia of the brain is sufficiently marked to produce coma, but not sufficiently marked to produce convulsions from the medulla oblongata.

d. Virchow's discovery that the ganglion cells are cretified beneath the seat of an injury to the skull, is also made by Fischer to support his hypothesis; he supposes that the nutrition of such ganglion cells is defective.

e. He accounts for the presence of blood in the urine by renal shock; in the kidney, too, vascular paralysis is followed by arterial anæmia and venous hyperæmia, which latter condition brings about hæmaturia by diapedesis.

f. To account for the presence of sugar and albumen, he refers to Bernard's experiments on the medulla oblongata. The more intimate connection is at present not manifest.

It must be confessed that Fischer's hypothesis is the only one which accounts for all the symptoms.

Of late it has been denied that there are cases of fatal concussion, in which no injury to the skull or soft parts, and, in fact, absolutely nothing abnormal, is to be found, save only abnormal blood distribution. Deville and Prescott Hewett are prominent in this connection, but it does not seem possible to overthrow the great number of trustworthy observations of this fact.

It has been universally admitted that *meningitis may ensue on simple concussion.* Of late, doubt has been thrown on this fact; Bergmann speaks with great reserve on this point, which he considers yet unsettled. Rose tells me that he does not remember, in all his large experience, a single case of meningitis, unless there was external or internal injury. At all events, it cannot be denied that meningitis may follow the group of symptoms which characterize concussion. In these cases the autopsy shows that something happened to the brain which was not able to modify the symptoms of simultaneous concussion.

Repeated experience of this kind has made most physicians very cautious in diagnosticating concussion. If coma be persistent, if it become even deeper, if sopor increase and the pulse become slower, some more deep-seated injury to the brain is highly probable. The appearance of symptoms pointing to meningitis is not surprising when we remember that some of the apparently simplest cases of concussion have afterwards turned out to be complicated; in fact, the certain diagnosis of this affection is impossible.

2. *Injury to the Soft Parts.*

a. Injury to the soft parts of the skull may give rise to meningitis, ragged and contused wounds and extensive lacerations being the most dangerous. Contusion is much more to be dreaded than multiple solution of continuity, for the former often gives rise to necrosis of the galea and infiltration of pus into the connective tissue between the galea and periosteum, and into the periosteum itself. Even if the galea does not become necrotic, it becomes so swollen that necrosis of the connective tissue is thus brought about. As these processes tend to progress, diffuse infiltration of the scalp, which of itself is a severe febrile affection and results in multiple abscesses, takes place. Then the periosteum becomes inflamed, often necrotic, and now nothing prevents suppurative ostitis being set up. If the process, on the other hand, limit itself, the result is merely more or less superficial necrosis, and the intracranial organs remain intact, though, if the necrosis be deep, meningitis may still eventually ensue. More commonly, however, the purulent ostitis extends inward, the dura becomes separated from the bone and infiltrated with pus, and thence the inflammation extends to the pia ; the inflammation can also take the other path and reach the sinus by means of osteophlebitis, or it may happen that everything at first goes well, and that very little of the tissue becomes necrotic; but suddenly erysipelas sets in, spreads, and exercises a most unfavorable influence on the granulations and the healing process ; the granulations become flabby, secrete thin ichorous pus and break down, and then the malignant suppuration spreads rapidly inward. The usual explanation is that the erysipelas has extended inward to the meninges ; but this explanation, though very attractive, needs further proof.

It follows from all this that no useful rule can be laid down as to the time which may elapse between the infliction of a wound on the external soft parts and the supervention of bad symptoms, referrible to meningitis. If the inflammation spreads steadily inward, meningitis may make its appearance after five or six days ; or, as we have stated above, it may be postponed till an incidental attack of erysipelas or necrosis of the bone supervene, as may happen at any time before the wound is healed.

In one case, which was under our own observation, meningitis supervening on a ragged and neglected incised wound proved fatal on the ninth day ; in a second case on the twelfth day ; and in a third case evidence of osteophlebitis was found. Bergmann reports a case in which erysipelas set in during the third week, and the ninth day thereafter the patient died of meningitis.

b. Injury to the soft parts and bone, but without perforation of the latter. Inflammation extends to the meninges in the same way in these cases as when the soft parts alone are injured. It is a matter, of course, that much depends on the character of the wound (whether there be contusion, comminution, a clean cut, etc.).

c. Injury to the head with an external wound, without any visible injury to the bone or the supervention of ostitis, but with splintering of the inner table.

A man fell from a wagon, but it was not possible to ascertain accurately on what part of his head he fell; he had the symptoms of concussion, and recovered. Three months later a new and acute brain affection came on and lasted some days, with diffuse pain in the head, delirium, and fever; he was treated antiphlogistically and the symptoms readily yielded. Still two months later he died at the end of the sixth week of genuine typhoid fever. A splinter was found detached from the frontal bone about one centimetre long and half a centimetre wide; the dura was somewhat thickened and perforated, but no pus was found. There was superficial yellow softening in the cortex (the second frontal convolution toward the apex of the frontal lobe) and perforation of the pia, but there was no trace of meningitis or pre-existent encephalitis. No cerebral abscesses.

Thus is shown that, provided the skull be not opened, splinters of bone may be tolerated, even if they perforate the meninges and cause superficial contusion of the brain substance. Fischer has also experimentally shown how tolerant the brain and meninges are of bits of bone or foreign bodies, provided only that there be no access to air. In the above repeated case the initial symptoms were simply those of concussion, and the injury to the brain was so situated as to give rise to no marked symptoms; but if it had been seated farther up it would undoubtedly have done so.

3. *Primary Meningitis as a Result of a Perforating Injury.*

It has already been stated above that purulent meningitis often supervenes on the symptoms of concussion, but that, in the great majority of these cases, after death complications are found which were the exciting cause of the meningitis, and it is a well-established fact that this latter may occur within an intact skull, though it is true that in the majority of cases there is perforation. In general the following injuries are to be borne in mind: linear incised wounds which penetrate the skull; penetrating contused wounds of the bone, with sharp or torn edges, and more or less crushing of the bone; wounds attended with loss of substance or with detachment of a bit of bone; severe contusions or fractures with or without perforation, from bullets or other cause, and in this class decomposing blood-clots play a great part; all sorts of fractures of the skull where a spiculum or a funnel-shaped group of spicula project inward, and give rise to all manner of lesions of the brain and its membranes; perforation or contusion of the meninges; injury to and disorganization of underlying brain substance.

The inflammation which is thus set up and favored by these various causes supervenes, sooner or later, on groups of symptoms, *which do not belong to commencing meningitis, but to generally disordered function of the whole brain or to the particular lesions which have been mechanically inflicted on the brain and its membranes.* These groups of symptoms go by the names of *compression* and *contusion* of the brain, and we will now proceed to devote a little space to their discussion:

Compressio cerebri—compression of the brain.—The above-mentioned injuries to the bone are often followed by the symptoms of compression of the brain, in that either direct pressure is made on the brain by depressed fragments of bone, or that a clot is formed between the dura and the bone. It is well known that elevation of the depressed fragments may be followed by the disappearance of the symptoms of compression (Bergmann, p. 91), and sometimes the symptoms disappear spontaneously and gradually without interference; in these latter cases the only possible supposition is that the clot has been reabsorbed. In some rare cases foreign bodies have given rise to compression. As soon, again, as an inflammatory process consequent on an injury has been set up in the skull, more or less marked evidences of compression will make their appearance. We have already discussed in full a peculiar form of compression, that resulting from an acute effusion into the ventricle; and among other causes we will enumerate hypertrophy of the brain, general œdema of the brain, enlargement of the brain caused by extravasation of blood or tumors, and finally, modification of the capacity of the skull by affections of its bony covering.

It is not easy to differentiate between the symptoms of compression and concussion; in many cases it is impossible, and we must temporarily rest satisfied with the diagnosis of disordered intracerebral circulation; in other cases it is possible to diagnosticate compression, inasmuch as the general symptoms of disordered circulation are associated with special indications of its cause. The most striking indications are:

a. In cases in which compression takes place gradually by virtue of an intracranial effusion of blood, there is a certain lapse of time between the infliction of the injury and the impairment of consciousness, and this lapse of time is occupied by certain brain symptoms which may be characterized as symptoms of active congestion—restlessness, noise in the ears, dancing spots before the eyes, flushing of the face, strongly pulsating carotids. Then vertigo and nausea are superadded, and soon after actual vomiting. Next come confusion of ideas, and finally headache and sopor. The latter does not seem to occur unless the capacity of the skull be diminished, and hence, already before its occurrence, the intracranial pressure must have become decidedly increased. It is easy to see that spontaneous hemorrhage (apoplexy) may have a similar commencement, the symptoms depending on the size of the ruptured vessel and the seat of the hemorrhage. If the vessel be small, a longer time is required before the necessary amount of pressure is brought about than if it be large; but the initial symptoms are the same. *Unconsciousness appears as soon as commencing general intracerebral pressure compresses the capillaries of the cortex.*

b. Pain in the head.—Compression of the brain always leads to pain in the head, doubtless due to pressure on and tension of the dura, which is extremely sensitive, as are not the pia and underlying portions of the brain.

c. Convulsions.—These are a very rare symptom in uncomplicated compression, and partial are more common than general convulsions. This must be due to the very gradual occurrence of the pressure, which, if it occur suddenly, as it sometimes does in spontaneous apoplexy, may be attended by convulsions. The usual cause, however, of traumatic compression is effusion of blood between the dura and the skull, and this must take place gradually on account of the rigidity of the opposing parts. Moreover, Pagenstecher and Bergmann have confirmed this experimentally; when the former allowed the pressure to rise gradually no convulsion took place, and the latter noticed in a child with meningocele that sudden pressure produced immediate convulsions; but gradual refilling, none.

d. The respiration is slow, regular, deep, and snoring; but gradually the intervals become longer and unequal—irregular respiration. If death ensues, respiration becomes gradually paralyzed.

e. The pulse becomes slow as the pressure increases; later it becomes irregular and then rapid, and sometimes is both irregular and rapid at the same time. These symptoms are the same as in artificial anæmia of the brain and medulla, though sometimes the pulse is first rapid and later slow (Landois).

Leyden considers this phenomenon in compression to be unquestionably due to irritation of the vagus when it coincides with a rapid pulse, but to paralysis of the vagus when it coincides with a slow pulse: if the vagus be cut while the pulse is slow, it rises immediately in frequency, and subsequent variations of pressure have no effect at all upon it. Thus, under these circumstances, the behavior of the centre of the vagus is precisely similar to that of other motor centres—its excitability is increased before its functional activity is lost.

f. If there be marked general compression, it is but seldom that the pupils are not dilated, and that their power of reaction is not gradually lost; but if the compression be partial, the pupil of the compressed side is alone dilated, and that of the other side is of medium size. It has been experimentally demonstrated that an initial contraction of the pupil of the affected side takes place; simultaneously the ball is rolled upward, the lids close, and sometimes nystagmus has been noted. As the pressure increases the pupils dilate; sometimes the pupil on the side of the compression is the first to dilate, while the other is of medium size. Up to this point the symptoms of concussion and compression have been so similar that in some cases a positive diagnosis cannot be made; but we now come to a few indications of the latter condition which facilitate diagnosis. More than once concussion has been diagnosticated, and further development of the symptoms has necessitated a change of view.

g. Unilateral paralysis of the extremities, commencing as paralysis of motion and impairment of sensation, but running into complete paralysis of both; simultaneous sopor, more or less marked. This is evidence that local is superadded to general increased pressure, *i.e.*, the condition which underlies the generally increased intracerebral pressure has reached such a point as necessitates abolition of function in the compressed portion of the brain. In these cases there is usually a large extravasation between the dura and the bone.

h. Fundus of the eye. If the pressure be considerable, the symptoms of choked-disc will be present. (The reader is referred to the discussion of this point under Tubercular Meningitis.)

i. In experimental investigations fever is wanting, the temperature either remaining normal or falling steadily up to the time of death; but in the human subject compression is often attended by moderate elevation of temperature, though only moderate. We have never known the temperature to be as high as in meningitis of the convexity.

Many observers have remarked that the general symptoms of compression are due to mechanical anæmia of that organ which is most sensitive to a withdrawal of nutrient blood—the cortex cerebri. The most recent experiments on this point go to show that, up to a certain point, the escape of cerebro-spinal fluid into the spinal canal depends on the elasticity of the ligaments of the vertebral column. This elasticity having its limits, the final result must be cortical compression, preventing the unimpeded circulation of arterial blood, a condition which is demonstrable *post-mortem* in cortices which were subjected to general pressure from within (hydrocephalus). This condition may be of any conceivable shade of intensity. A degree of compression which is not sufficient to arrest the circulation in the cortex may, owing to the extreme sensitiveness of the dura, be sufficient to give rise to decided subjective symptoms (headache), though the sensorium be spared. But as soon as the capillary circulation is so impeded that the normal interchange between the blood and the tissues no longer takes place, general impairment of cerebral function results. Attention has already been called to this in connection with those disorders which result from extreme hydrocephalus (impeded flow through the small arteries, stagnation of blood in the capillaries). If the effects of general cerebral pressure are to be regarded in this light, the question remains to be answered, whether partial cerebral pressure has any influence on the immediately underlying portions of the brain (depression of the skull, traumatic extravasation of blood); in other words, whether in general cerebral compression symptoms of local trouble can be referred to the portion of the brain where the pressure is at its maximum. Leyden and Bergmann, among others, have shown that in partial compression the pressure is decidedly diminished at some distance from the seat of the lesion. Now, if this fact apply only to compressed and not to disorganized brain tissue, the only conclusion to be drawn from it is that the affected portion is completely shut off from the circulation by maximum compression of the capillaries, and that that portion of the brain is, consequently, physiologically dead. But Bergmann has insisted especially on the fact that the general tension in the cavities of the skull and spinal canal is increased even by local pressure.

If the central organ of the nervous system be cut off from its circulation, or if a cardiac affection prevent the continuance of the normal blood current, it is the cortex which first shows a loss of function. Under these circumstances (general vascular spasm, sudden feebleness or paralysis of the heart), general convulsions, which have been proved to originate from the medulla oblongata, appear, and that which merely suspends the function of the cortex proves an intense irritant to the medulla. When anæmia has been brought about experimentally—for instance, by gradual bleeding to death (Kussmaul and Tenner)—death is preceded by swooning and loss of consciousness, but not by convulsions. Thus, rapid fluctuation of nutrition excites the great ganglia, and gradual fluctuation does not; but the effect on the cortex is the same in both cases. This fact is opposed to the other explanation of the convulsions, viz., if the arteries which supply the brain be ligated, the reflex inhibitory centres of Setschenow are paralyzed, and thus free play is given to spinal reflex action. If we now apply these considerations to compression of the brain, we must refer the chief symptom, sopor, to mechanical impairment of circulation in the cortex, and the great variety of convulsions to the slowness with which the consequences of the compression are transmitted to the great ganglia.

Surgically speaking — and traumatic meningitis comes eminently within the domain of surgery—cerebral compression is due to the manifold forms under which extravasation of blood in the cavity of the skull takes place, to depression of the bone exerting direct pressure on the brain, and, finally, to the following conditions: *diffuse suppuration of the pia, superficial encephalitis and diffuse suppuration of the brain, abscess of the brain.*

Traumatic extravasations within the skull originate generally from rupture of the middle meningeal artery by a sharp edge of fractured and depressed bone. If one consider but for a moment how the artery lies in a deep furrow, it is an easy matter to understand how sudden depression and re-expansion of the bone may tear the artery. Similarly, extravasation takes place between the bone and the dura, from rupture of the sinus.

A severed artery will, of course, continue to bleed until the external and internal pressures are equalized, and the largest extravasations always originate from the middle meningeal artery. Sometimes they are of sufficient extent to separate the dura almost completely from one hemisphere. According to Pagenstecher, the mean which is necessary to give rise to compression is 2.9 per cent. of the capacity of the skull; the maximum is 6.5 per cent.; though, according to his experiment, these estimates are subject to considerable fluctuations. We have noted an extravasation which weighed two hundred and six grammes, thus decidedly overstepping Pagenstecher's limits both in volume and weight, though it was found in a case in which senile atrophy of the brain was very marked.

The symptoms of such a hemorrhage are not difficult to recognize, inasmuch as they correspond to pressure which is developed gradually, a certain period of time elapsing between it and the trauma. In uncomplicated cases the injury is followed by no severe immediate consequences. These do not appear for a certain length of time (one-sixth, one-quarter, one-half hour—at most two hours), during which the blood is accumulating. These symptoms are: noise in the ears; pain in the head, gradually increasing in intensity; giddiness, vomiting, fatigue, listlessness, sleep, sopor, snoring respiration, slow pulse, dilated and feebly reacting pupils (see the above-mentioned exceptions), and, finally, coma. Such a condition may last a long time before resulting in death, may be only partially developed, or may even be attended by fluctuations, which are doubtless dependent on temporary remissions of the active congestion (treatment). Hemiplegia of varying degree constitutes the chief diagnostic sign. If symptoms of compression appear first several days after the trauma, they cannot be referred to a rent in the meningeal artery or the sinus.

Although the affection is often clearly indicated by marked symptoms such as those above mentioned, unfortunately this is not always the case, for *consciousness is often lost immediately after the injury.* To the symptoms of compression are superadded those of concussion; or, may be, contusion of the brain forms a still further complication. In such cases not even hemiplegia is diagnostic; for, although in uncomplicated compression we ascribe this symptom only to fatal crowding of the blood out of a superficial portion of the brain, in cases of contusion, of course, the same thing takes place. This leads up to the consideration of contusion of the brain. Experience teaches us thus much, that cases occur in which we cannot distinguish between concussion, compression with extravasation of blood, and contusion.

Hemorrhage into the subdural space may occur independently, provided that a trauma rupture those veins which connect the subpial space with the longitudinal sinus (see above, under Pachymeningitis Hemorrhagica). Such rupture of the veins has been observed without any injury to the bone whatever, and, in like manner, the longitudinal sinus may be laid open in such a way as to give rise to hemorrhage into the subdural space. Finally, both the dura and the pia may be torn, and thus the subdural space be occupied by portions of both extra-dural and subpial extravasations.

Hemorrhage into the subpial space is a concomitant of every superficial contusion of the brain. Blood is infiltrated into the network of the pia, and can be detached together with it; but the volume of these extravasations is never sufficiently great to give rise to compression.

Contusio cerebri.—The relation between this condition and traumatic encephalitis compels us to give it brief consideration. In this case too, for some time back, the authorities have drawn a familiar sketch of the condition as characterized by peculiar symptoms, although Dupuytren, who was the founder of our conception of contusion of the brain, from the very beginning followed a more rational course. Of late this condition has been freed from the mysterious bonds of a dynamic influence (accompanied by characteristic symptoms) on the brain as a whole. To-day it is regarded merely as the physiological sequence of a sum of the different injuries which take place in contusion, and the consequence of which varies with time and seat.

Contusion takes place—

a. Whenever the skull undergoes considerable change of form suddenly; and it may occur without any injury whatever to the bone, since the cerebral substance offers less resistance than its bony covering.

b. Injury to the skull, without perforation, but with splintering of the inner table and superficial injury to the brain.

c. Injury with depression, and with or without splintering.

d. Injury by projectiles, and lodgment of the same in the cavity of the skull.

The very nature of all these injuries is such that the greater part of them perforate and lay open the cavity of the skull, and consequently constitute the chief source of acute meningitis or meningo-encephalitis. The latter condition may supervene on contusion without injury to the skull, though not so commonly as when there is perforation.

The seat of the contusion of the brain may be either immediately under that portion of the skull where the injury was inflicted, or on the opposite side of the brain, or in both of these places at the same time. According to Bergmann, if the head rest on some hard body, and a dull and heavy blow be inflicted on the opposite side, the brain suffers more on the former side than on the latter. By reason of the fact that most injuries of this kind are inflicted on the superior surface of the skull, contusion is more common on the convexity than on the base. Bergmann lays down the very interesting law that contusion is more common at the base than on the convexity, if the blow be very heavy and dull, as from a fall. But, if we have to deal with injuries which are very limited in extent, the convexity is more likely to suffer. It is, moreover, evident that, other things being equal, the more delicate portion of the brain, the cortex, in which the ganglionic element predominates over the fibrous, should offer less resistance than those portions, as the medulla, the pons, and the peduncles, in which the fibrous element predominates; but there is no portion of the brain which has not been disorganized by force exerted from without. Meningeal hemorrhage is an almost constant concomitant of contusion, and separation of the dura, and formation of a clot between the dura and the bone, are not very rare, though more rare than in the affection discussed in the former section. Bergmann considers hemorrhage so constant that he counsels us to avail ourselves of it for diagnostic purposes in doubtful medico-legal cases.

Contusion of the brain is always associated with solutions of continuity of its constituent elements. Thus, not uncommonly contusion is evidenced by scattered or grouped capillary apoplexies through the substance of the brain, but more marked in the cortex. Formerly it was doubted whether these capillary extravasations were to be ascribed to contusion, some of the older authorities attributing them to concussion, and some even seeing in them the cause of the symptoms in concussion. But of late it is universally admitted that they are dependent on contusion, and do not occur in pure concussion.

If the injury be confined to a very limited portion of the brain the capillary hemorrhages are not diffuse, but may present an appearance very similar to that of a hemorrhagic infarction. Just as some infarctions leave the tissue of the brain macroscopically intact, so may contusion result in a larger or smaller collection of extravasations, capillary in size or even larger, which are thickly aggregated in the centre of the mass and become more discrete toward the periphery, the mass having no well-defined edges, but being gradually merged into the surrounding healthy cerebral tissue. The centre is always tinged more or less deeply, being of an uniform dark red or brownish shade. In other cases the extravasations are much larger and more closely aggregated, and are accompanied by laceration of the brain tissue, which is appreciable to the naked eye. This laceration is generally superficial, and is caused by a marked depression or a number of loose splinters. The centre of the mass may thus consist of a thrombus of coagulated blood, which is surrounded by smaller extravasations, becoming capillary toward the periphery. As far as the capillary apoplexies extend the whole spot is deeply stained with the coloring matter of the blood, and without clearly defined edges is merged into normal tissue. A still higher degree of laceration is brought about by perforating projectiles, fragments of shells, etc. Large portions of the brain may be thus disorganized and ground into a reddish mass which consists of blood and brain tissue. The transition into normal tissue is marked by the absence of capillary apoplexies and staining by the coloring matter of the blood. Of course, in greater or less degree the meninges almost always share in the laceration and bruising.

The diagnosis of *contusion* has been very thoroughly and clearly discussed by Bergmann, who agrees with other authorities in calling particular attention to the fact that the contusion is very rarely uncomplicated, but nearly always associated with the symptoms of both concussion and compression, since the conditions under which they occur (jar and extravasation of blood) are nearly always present. In the diagnosis of injury to the brain he recognizes but one rational sign as of value, viz., disordered function which can be localized in the brain. This is, of course, more easily recognized in the case of paralysis of a particular nerve—the facial nerve, for instance—than in the case of convulsions or contractures; convulsions, as is well known, do not assist us at all in localizing a lesion unless they are confined to particular groups of muscles. The first condition which Bergmann considers essential to diagnosis is a careful distinction between the more general symptoms caused by disordered circulation, and those symptoms which indicate a local lesion. The second condition is, from the localization symptoms to estimate the seat and intensity of the lesion. When we consider that in many cases it is utterly impossible to comply with the first condition, the great difficulty in localizing the lesion with the assistance of contradictory indications, often very few in number, becomes immediately evident.

a. Unilateral paralysis supervening on an injury with the symptoms of compression is not diagnostic of contusion unless this condition can be made out from an examination of the wound itself. If there be an extravasation from the middle meningeal artery between the dura and the bone, its effects are general and local: general, in that it leads to a general increase in the intracranial pressure, that is to say, to compression; local, in that the hemisphere of the affected side is more compressed than its fellow. Thus occurs in many cases complete or partial paralysis of the opposite side. *The symptom is a source of error in that it may be caused either by an extravasation which gives rise to compression, or by contusion of the brain.* There is no refuge from the dilemma in the case of compression, and the further development of the symptoms does not always clear up the diagnosis. There is but one condition which renders a positive diagnosis possible: if, owing to the nature of the injury, no extravasation of moment takes place, but a motor tract of the brain is injured (superficial motor centre), and paralysis of the opposite side ensue, contusion can be diagnosticated with certainty.

b. General convulsions are very rare in this class of injuries, though we have noted them once in a rapidly fatal case in which the clot was situated around the medulla oblongata, and there was fracture of the base; not that they may not occur in connection with another sort of injury, direct pressure of a clot on the medulla being a great rarity. Convulsions have no diagnostic value in this affection.

c. Unilateral convulsions which involve the whole side, or which, beginning with the facial, for example, involve next the muscles of the eye and the hypoglossus, next the territory of the spinal accessory, and, finally, the arm and leg, by which time they have left the face, are of far more value. This form of convulsion is not common, and has been noted by us in only one case, in which there was contusion of the left frontal and parietal lobes. The disorganization was slight, but the cortex was dotted with apoplexies over a large extent.

d. Of still greater value may be convulsions which are localized in special groups of muscles. Since Hitzig has taught us that definite motor functions belong to definite portions of the brain, we have become enabled to localize a number of pathological motor phenomena. It is true that we oftener have occasion to localize a brain lesion by the aid of complete or partial paralysis, or a condition of deficient muscular sensation, than by the aid of convulsions, though cases are on record of circumscribed convulsions, in which the lesion has been correctly localized. It is to be borne in mind that those fibres which are still intact and end in the injured part, become greatly irritated by the subsequent local inflammation, and thus the ultimate cause of the irritation remains undetermined.

It is to be remarked that there is no fixed relation between convulsions and contusion of the brain. In a case of large extravasation between the skull and the dura we have seen clonic spasms in the opposite leg, although the supposed muscular centre of the inferior extremities was neither directly compressed nor disorganized, the extravasation being seated too far back to admit of this taking place. Moreover, convulsions supervening on an injury of the cortex may stand in no manner of relation to this latter, for the reason that neighboring fibres may be irritated, and thus the localization be invalidated. The sequence in the convulsions which is sometimes observed leads us to suppose them to depend upon periodically increasing and diminishing irritation, which affects certain groups of fibres, only to leave these again for others; such irritation can hardly be due to anything else than a circulatory, and hence transitory, disturbance. Finally, convulsions, to be of any diagnostic value, must make their appearance very soon after the injury; otherwise they are open to the suspicion of being due to meningitis.

e. Convulsions in paralyzed parts.—The power of voluntary movement may be lost in a part, and still clonic spasms may break out in it, and reflex movement be called forth by strong irritation. This anomalous condition is only to be explained by loss of function in the centre of volition of the cortex, while the motor fibres of the corona radiata, which lead to it, still retain their excitability.

f. Aphasia.—As partial meningitis may destroy the function of considerable portions of the cortex, so we meet with cases of traumatic brain trouble which are characterized by aphasia, and in every one of these cases which come to autopsy the injury was of the left hemisphere (see Bergmann, p. 273). A case of fall on the head has come under our own observation, in which aphasia, without any other form of paralysis, persisted for fourteen days; also in a case of fall from a tree, on the left side of the head, paralysis of the opposite arm and leg, and aphasia, persisted till death, and the autopsy showed the remains of a contusion of the left island of Reil, and yellow softening in large extent. In general, no case of aphasia following injury or disease of the right side has come under our observation.

g. Lesions of the basal nerves.—These are of great value in the diagnosis of fracture of the base of the skull, and under some circumstances may be of assistance in recognizing contusion of the base of the brain. Seeing that they are chiefly valuable in connection with the former of these conditions, which does not come within the scope of our subject, we will pass them over, and refer to Bergmann, p. 221.

We have said enough to show that the injuries which lead to meningitis are very various, but that they have a more or less common factor in that they allow air to enter the skull. It results from the above that the primary affections which may be followed by primary and rapid meningitis are manifold: wounds of the soft parts and bone, with simple and sure signs of general, and often also of partial compression (traumatic extravasation between a fractured portion of the skull and contused brain), but in which the diagnosis of contusion is always very doubtful; wounds of the soft parts and bone, with signs of peripheral injury to the cortex, but without those of compression; contusion, without extravasation of moment; wounds of the soft parts and bone, with signs of concussion, with or without superficial disorganization which can be localized. Let it only be remembered that the absence of peripheral symptoms, paralysis, spasms, etc., and the absence of aphasia, do not exclude contusion.

There are two chief conditions which, under such circumstances, may favor the occurrence of meningitis, and on these conditions modern surgery lays due weight. The first and most important consists in decomposition, which rapidly takes place on the surface of the wound itself, and which is analogous to what takes place in all contused wounds. The secretion from the wound, which is rapidly poisoned by atmospheric germs, bathes and infects the contused and lacerated pia, the anatomical structure of which is most admirably adapted to spread the inflammation far and near. It is chiefly through the rich supply of lymph spaces in the pia that poisonous matter gets to distant points; but decomposition also takes place in the extravasations, which are always present in the pia, and this alone is enough to set up inflammatory disturbance. Fischer has shown experimentally the share which belongs to splinters and sharp edges of bone in the etiology of meningitis. There is still some room for doubt whether a brain enclosed in an intact skull moves with the systole of the heart and the respiration, although it is an indisputable fact that such movement does take place when the skull is open. In the case of perforating wounds of the skull, Fischer sees in every projecting edge of bone, in every splinter, a source of meningeal inflammation, since the brain and pia rub against it with every movement of the former. Fischer has shown experimentally that such a sharp projection will be long tolerated, provided that the skull be not opened; just as we sometimes find splinters detached from the inner table, which have torn the dura and pia, and caused superficial yellow softening, but no further damage. Nails which were driven into the intact skulls of animals did not set up meningitis; but when a nail was inserted at the circumference of a perforation by the trephine, meningitis was set up in a short time. This justifies us in regarding splinters and sharp fragments of bone, with perforation, as excitants of meningeal inflammation. The experience of Rosenthal is similar: he considers friction of the inferior surface of the brain and the base of the skull responsible for meningitis when the spinal canal is opened by trauma.

The length of time which elapses between the injury and the unmistakable appearance of meningeal symptoms varies, according to circumstances, from one to ten days. If meningitis appear in the second week, it may still be considered primary. There are many factors which concur in bringing about this variation. The nature of the injury, the size of the perforation, the degree of splintering and contusion, the size of the perforation of the meninges and the extent of superficial contusion of the brain, and, finally, how many and what manner of atmospheric germs become entangled in the open surface. All these circumstances are of importance in determining the acuteness of the meningeal inflammation. The germs, indeed, are of such importance that some authorities find it *difficult to credit the occurrence of purulent meningitis in contusion of the brain, if the skull be intact.* Bergmann does not consider that we have sufficient facts at our disposal to warrant a positive answer to this question, and other observers hold the same opinion. A number of cases have come under our own observation in which no suppuration whatever took place in spite of the presence of splinters from the inner table. Notwithstanding this, we must, relying on trustworthy observations, concede the possibility of the occurrence of meningitis, although the skull be intact.

4. *Meningitis Secondary to Traumatic Ostitis (Perforation of the Skull).*

We have already in a preceding section touched on this form of meningeal disorder, and insisted on the fact that meningitis, which is secondary to traumatic ostitis, is always later in its appearance than the primary form. Suppurative ostitis generally occurs at the end of the first or beginning of the second week, but may occur much later. The more extensive the contusion of the bone and extravasation in the diploë, and the more intense the primary decomposition on the wound, the more extensively and rapidly does suppuration spread in the diploë; sometimes it extends actually through the bone to its external or internal surface. The most favorable result which can then be hoped for is necrosis and gradual exfoliation of the inflamed portion of bone.

During the acute period of the ostitis (from seven to fourteen days on an average after the injury) the patient is in constant danger of meningitis, even if he has the rare good fortune to escape without contusion of either dura, pia, or surface of the brain. In any other case meningitis is almost sure to follow. It is readily seen that the period of time which elapses before this form of meningitis is set up is no fixed and definite one. We saw that primary meningitis generally occurs in from one to ten days, meningitis secondary to ostitis in from seven to fourteen or eighteen days; new cases which arise between the seventh and tenth days may be classed under one head as well as under the other. This is no more than is to be expected from the nature of the case, since direct infection of the pia from the wound may set up simultaneously meningitis and purulent ostitis, and hence the origin of the former remain obscure. At this point the primary and secondary forms cannot be clearly distinguished. Meningitis secondary to ostitis is sometimes the result of suppuration in a thrombus of the sinus. No general conclusions can be drawn from the period of time which elapses between the trauma and meningitis, for it varies within the widest limits; but to this form the appellation of "secondary meningitis" is chiefly applicable.

5. *Decomposition in Clots between the Skull and the Dura the former being perforated.*

All surgeons admit the existence of this form of meningitis (vide Bergmann, p. 249).

Primary decomposition takes place in the wound, and as a consequence of this the clot between the bone and the dura is infected. The detached dura, which is perhaps already necrotic, becomes soaked in foul secretion, which extends to the pia and then sets up immediately purulent inflammation; or else the dura has not become necrotic, but purulent inflammation is set up on both its surfaces from the mere contact with these foul secretions. It is due to the varying degree of acuteness, that sometimes purulent inflammation is confined to the outer surface of the dura, while the inflammation on the inner surface is adhesive in character and thus protects the pia. This can only happen when the portion of dura involved in the inflammation is very limited.

6. *Tardy Meningitis during the stage of Traumatic Necrosis.*

This form of traumatic meningitis is one and the same with that which has already been briefly considered, when the pia becomes involved from chronic inflammation in neighboring bone. The period of time which elapses between the injury and meningitis, in these cases varies enormously. We have known secondary meningitis to recur, in a case of perforation of the skull by a piece of falling wood, in the twelfth week.

Pathological Anatomy.—It is impossible to describe here all the appearances which may be the result of traumatic meningitis. Manifestly, the most important question is this: whether the pia alone is involved in the macroscopic inflammatory process (microscopic suppuration always takes place in the cortex, even if there be no disorganization of moment), or whether, as is more frequently the case, at a contused point superficial encephalitis with diffuse infiltration and pus has been set up in addition to the meningitis. If this latter be uncomplicated, the following are the important points:

Lepto-meningitis begins and is most intense at the seat of the injury, whenever it spreads rapidly in all directions. We often see the inflammation at that stage when at and near the seat of the injury the pia is full of thick layers of pus, which, at a little distance, is pretty closely confined to the course of the larger vessels. The microscope, however, shows us that the inflammation has extended to portions of the pia which appear perfectly normal to the naked eye.

Sometimes suppuration is confined to the outer surface of the pia—that is to say, a thin layer of fibrinous exudation, very rich in pus cells, is found on the pia in the subdural space. A greater or less degree of internal purulent pachymeningitis is always associated with this condition, but there is never any great accumulation of pus in the subdural space.

A peculiar character is often imparted to suppurative meningitis by hemorrhages into the pia which rapidly break down. The coloring matter of the blood is diffused widely in the neighboring parts, and both the pia and cortex may get a shade of red or brown, which is evident in spite of the suppuration.

The seat of meningitis has much to do with its extent. If the seat be the convexity of one side, the patient very often dies before the inflammation has reached either the base or the convexity of the opposite side. The case may be similar when the base is first involved, though in this case the inflammation seems to have a tendency to spread backward to the medulla oblongata and the cervical portion of the cord.

The choroid plexus often becomes involved as well as the pia, and then ensues modification of the intraventricular fluid, both quantitatively and qualitatively. In traumatic meningitis, too, the fluid may be increased in quantity and opaque, and may, even in rare cases, give rise to compression; though generally the increase is not very marked, and depends on an inflammatory condition of the plexus, which is less intense than at the point where the meningitis originated. Exceptionally, the exudation is rich in flakes of fibrin, and sometimes the plexus is coated with a pyo-fibrinous deposit. An exudation consisting of pure pus is also rare, though it does occur. We must doubt whether uncomplicated transudation into the ventricles of traumatic origin occurs. Bruns, on the authority of Abercrombie, says that it does occur, but the observations of the latter are very far from showing it to be the result of traumatic meningitis.

Meningitis accompanying contusion of the brain is usually, indeed almost always, combined with superficial encephalitis, which results in suppuration. This suppuration may be very great, and the collection of pus between the pia and the sound portions of the brain astonishing in amount. It is covered by torn pia and rests on a ragged base of brain substance in which inflammatory action has been set up, but has not, as yet, advanced to disorganization.

Thus it is shown that in cases of severe contusion meningitis and encephalitis coexist—a fact which has led many writers to fuse the two affections into one. Though the etiology of the affection would lead us to suppose that meningitis alone is often found, microscopical examination of the cortex shows us that we should be in error. Unquestionably it is only in the very commencement of purulent meningitis that the cortex is free from microscopic changes. Our surgical experience—perhaps too limited to settle the question—goes to show that, if the meningitis be considerable in extent and intensity, not only do we have all manner of circulatory disorders which vary according to the stage of the affection (initial hyperæmia, terminal anæmia), but also emigration into the cortex of the red and white corpuscles; the ependyma also is, as in tubercular meningitis, infiltrated with pus-cells, and emigration, in varying degree and intensity, is strongly marked along the course of the vessels. Our investigations have convinced us that acute changes take place in the ganglion cells (a granular condition of and loss of continuity in the protoplasm), a fact which has hitherto attracted but little attention, and which doubtless depends on the acuteness which characterizes the affection in question. This condition of the cortex is analogous to that acute and diffuse cortical encephalitis, the gross appearances of which are very similar, and which is clinically a maniacal form of dementia paralytica.

Symptoms.—We have already shown that in many cases meningitis and peripheral purulent encephalitis coexist; hence is it most difficult in any given case to distinguish the two affections. Indeed, cases of this kind occur which are not distinguishable from uncomplicated meningitis by a single symptom of value, unless, indeed, the cortical lesion involve a portion of the brain which is the centre of a special function (island of Reil, frontal lobe). In the latter case we are apt to have those symptoms which have been already specified as of value in the diagnosis of contusion. Even these, however, are not absolutely trustworthy, since at least one of them, aphasia, has been observed by us in uncomplicated meningitis, traumatic as well as non-traumatic. This is a case in point: a heavy beam fell on the left parietal region, causing fissure, depression, and signs of concussion, which disappeared in the course of twenty-four hours; but the patient was still somewhat out of his head, though his articulation was perfect. After four and a half days signs of intense irritation of the brain came on; high fever and aphasia; sopor and coma soon supervened, and soon afterward death. The autopsy showed purulent meningitis over the left hemisphere and fossa of Sylvius. The surface of the brain itself appeared healthy to the naked eye.

We have now shown that various symptoms precede those of meningitis itself, and these have been already considered in sufficient detail. It may be as well, however, to insist once more on one point, viz., that the results of the inflammation are both anatomically and symptomatically the same in contusion of the brain as in fracture of the skull without contusion of the brain; the same in perforation of the skull without contusion, but with extravasation of blood, as in a like condition but without extravasation. The length of time which may elapse between the injury and the commencement of meningitis in its various forms has been already discussed.

The commencement of the symptoms is essentially modified in that in one case the meningitis attacks a patient who is already in a condition of sopor, whose pulse is slow and weak, whose temperature is normal or but slightly higher than normal (concussion, compression); in a second case a patient who is conscious, and in whom the only morbid signs consist in some sort of wound on the head, the earliest symptoms of commencing inflammation, and a correspondingly elevated temperature; in a third case a semi-conscious patient, in whom symptoms of local lesion (paralysis, partial convulsions, clonic spasms, aphasia) give almost positive proof of partial disorganization of the brain. In cases of concussion with doubtful injury to the brain, the determination of the period at which meningitis began is rendered still more difficult by the appearance, on the third or fourth day, of symptoms of *active congestion. There may be no dividing line between these symptoms and such as justify us in assuming the existence of meningitis*, and there is nothing to mark the time at which congestion ceased and meningitis began. Every physician is rendered anxious by symptoms which approach so closely those of meningitis.

The signs of congestion are restlessness, jactitation, the patient tries to leave his bed, clutches at his head or at the air, wrinkles his forehead, distorts his countenance, throws around everything within his reach, especially any dressings which may have been applied to his head; busies himself with his genitals, and resists restraint or interference; sometimes he is violently delirious and raves. Belching, vomiting, grinding the teeth, yawning, expectoration, congestion of the face and conjunctivæ, also may occur.

Vascular excitement, *the pulse becoming fuller* and cardiac contraction more forcible. The pulse increases in frequency, though even if a slow pulse becomes still slower, we have no ground for encouragement if there be compression. *It does not indicate meningitis*, but active congestion must intensify the compression which already exists. Patients who are conscious complain of severe pain in the head, giddiness, buzzing in the head, noise in the ears; they are sensitive to external impressions and shun the light; often they are very irritable and peevish. *The temperature* is very variable, and up to the present time investigation has been unable to establish any fixed rule in regard to it. At least, *a rise of three or five quarters of a degree C.* (*from one and a third to two and a quarter degrees Fahr.*) *cannot be regarded as indicative of meningitis.*

All these symptoms may be transitory, and a diagnosis impossible before the termination of the affection; this is especially the case in cases of concussion, in which the condition of the brain is open to doubt. In many cases, however, symptom is so added to symptom that there can be no doubt as to the diagnosis, and in other cases the course is so rapid that even death occurs before the diagnosis can be made.

The indications of meningitis are: *Long duration* of the above enumerated signs of congestion—several days. *Reappearance* of the same after remission. *Persistence of the fever*, even in the morning; high fever is always of unfavorable import. Bergmann correctly remarks that a temperature of more than 39° (102.3° F.) is of positive evil omen; we have known it to rise above 40° (104° F.). *Persistent violent delirium.*

Persistence of slow pulse, or excessive frequency in the pulse; sometimes an early indication of paralysis.

As in the other forms, so in this, the indications that the affection has become developed may be divided into two classes: those connected with irritation, and those connected with paralysis, of the brain. Such a division is never absolutely trustworthy, and, indeed, is even less so in this form than in those the course of which is less rapid. The above-named signs of congestion are often also those of inflammation, and any one who counts upon a marked stage of irritation will be disappointed, inasmuch as the signs of cerebral paralysis are developed with the greatest rapidity, and it can truly be said that of all the forms of meningitis the traumatic form is the most deceptive in this respect.

It is matter of experience that the symptoms of irritation which belong to meningitis are the same as those which we have already enumerated as belonging to initial congestion. Piercing and tearing pain in the head, sleeplessness, sudden starts as if from alarm, thirst, complete anorexia, etc. Consciousness is very often already impaired, but as long as a degree of consciousness persists, the patient is easily aroused. Often now he becomes increasingly delirious; his delirium is of the most variable nature, and often becomes very rapidly violent. Now supervene some symptoms of irritation which throw light on the diagnosis.

Clonic spasms, in special groups of muscles, doubtless occur in meningitis without primary contusion of the brain, and are dependent on microscopic changes in the cortex. Later in the course of the affection these spasms generally disappear, as, in fact, do all signs of innervation during the paralytic stage; the spasms may, however, return after some time. In cases of traumatic meningitis we have never known paralysis to be limited to those muscles which were affected with spasms.

Tonic spasm and contracture in a limb. This is to be regarded as a transitory phenomenon, inasmuch as it generally disappears on the advent of paralysis. We have never known it to be followed by limited paralysis. Tonic spasm of the muscles of the neck is a common and reliable indication; sometimes it involves all the muscles of the back, and even, in varying distribution, those of the extremities, so that the whole body is in a condition of tetanoid rigidity. General convulsions seem to be rare in traumatic meningitis.

Retraction of the abdomen rarely or never occurs in traumatic meningitis.

Constipation is always present; vomiting comes on sometimes sooner, sometimes later; thirst is great; the tongue is usually dry, often cracked and of a dirty brown color. We have already alluded to the rapid course of traumatic meningitis. Hence it may happen that the above-mentioned symptoms of irritation may be but faintly marked or quickly disappear, and the symptoms of depression follow so rapidly that, though the symptoms of both these conditions may coexist for a time, very soon the symptoms of paralysis predominate.

Mental excitement gives place to quiet and insensibility; the senses are blunted, or, in other words, the perceptive organs either lose their functional activity entirely, or, if it be retained for a time, it is greatly impaired. The sensibility of the cutaneous surface is diminished; if hyperæsthesia has been present, it quickly disappears; painful irritation of the skin gradually evokes merely reflex action. Delirium gives place to sopor, which becomes even deeper, and finally ends in coma. Now even motor symptoms of irritation disappear; rigidity of the neck last of all, though this also generally vanishes shortly before death. Gradually, as coma comes on, reflex movements also disappear, even those simple ones which originate in the cord and medulla oblongata. The latter retains its excitability longer than any other portion of the brain, longer than the cord, which is subjected to pressure in the vertebral canal. Finally, however, it too is paralyzed by generally increased intracerebral pressure.

Paralysis as well as spasm, which is followed by paralysis, stands in very peculiar relation to meningitis. It cannot be affirmed that paralysis never occurs in genuine traumatic meningitis, for if it be seated at the base, it quite commonly gives rise to paralysis of the cranial nerves.

Ptosis, *palsy of the abducens*, *partial palsy of the oculomotorius* (the pupils), *palsy of the facial*, *of the hypoglossus*, *of deglutition*, all are met with. We must only remember that isolated paralysis may depend on direct injury to the nerves themselves, as when it appears earlier than meningitis.

The above remark, that in pure meningitis limited paralysis does not seem to occur in muscles which have been the seat of clonic spasm, does not apply especially to paralysis of the extremities, since paralysis of a particular limb, and even hemiplegia, are sometimes met with. We do not know accurately in what portion of the cerebro-spinal axis the cause is to be sought. In general meningitis we have known hemiplegia to occur on the side opposite to the less diseased hemisphere. This was accounted for by an intensely inflammatory condition of the peduncle. We shall discuss the symptoms of local lesion which occur in encephalitis in connection with that affection.

As in other fatal meningeal inflammations, the gradual approach of paralysis of the medulla oblongata is indicated by modification of the pulse and respiration. The pulse becomes small, and rapidly increases in frequency; sometimes there is a rapid increase in the temperature, which also may rise after death; or, in some cases, fall during the agony. The respiration, which for some time has been slow and snoring, becomes toward the last rapid, superficial, and finally, intermittent. The appearance of the retina is the same as has been already described in connection with other forms of meningitis.

The usual termination of purulent traumatic meningitis is, unfortunately, death. In discussing the possible termination of the affection, we are met again by the old difficulty in deciding whether, in any given case which has terminated in recovery, suppuration really took place, or whether there was nothing more than intense congestion. Owing to the variability in the character of injuries, and of their primary effects, to individual peculiarity and its influence on congestive processes, to the impossibility, even anatomically, of distinguishing with certainty between congestion and inflammation, we consider an accurate differentiation impossible. Both parties may be right: those who maintain that traumatic meningitis is often recovered from, as well as those who deny that this is the case. Under favorable circumstances, doubtless, during its early stages, the process may take a favorable turn, but extensive purulent infiltration of the pia is fatal.

The *duration* of traumatic meningitis varies within wide limits. The most acute cases terminate in two or three days, and in some cases an irritative stage seems to be completely wanting, and symptoms of depression to come on immediately. Bruns lays down from eight to fourteen days as the usual duration; we consider this longer than the average. It appears to us a great mistake to attempt to fix an accurate duration, since it is often impossible to determine accurately the exact time at which meningitis began; Billroth also calls attention to this fact. He is of the encouraging opinion that in traumatic meningitis also no inconsiderable portion of the symptoms (fever—general manifestations) are due to that infection of the blood, which is sure to take place if the injury perforate the skull and allow access to the air. Thus, unquestionably, a quantity of poisonous material is formed, and may be as readily absorbed as in traumatic pleuritis or peritonitis. This theory explains many cases in which there is surprising discrepancy between the severity of the constitutional disturbance and the post-mortem appearances.

For *treatment*, see the end of the following section.

III.—Encephalitis.

We come now to consider circumscribed encephalitis, or rather, that form of it known under the name of "red softening," which has a tendency to lead to macroscopic collections of pus in the inflamed spot. The sclerotic forms will be described in another place. All the diffuse forms will be described in the following section, although one of them (diffuse encephalitis of the cortex) is closely related to local encephalitis.

It is not long since there was much discussion as to the signification and anatomical bearing of red softening or encephalitis. It was only after the expenditure of much time and labor that physicians succeeded in discovering the true pathologico-anatomical bases of the different softenings which are so frequently found in the hemispheres. At the present time, however, no one doubts the essential difference of the softenings which occur in connection with chronic changes in the vessels of the brain, from true encephalitic lesions. Here, again, Virchow's investigations have opened the way, though before him Carswell, and, among the Germans, especially Hasse, possessed perfectly correct views on the subject. A constant state of confusion, however, was maintained by the incomprehensible obstinacy of Durand-Fardel, who persisted in attributing all the softenings of the brain to an inflammatory origin.

Our standpoint to-day is as follows:

I. *Red softening and abscess of the brain are true inflammatory lesions of the cerebral substance.* In the brain the inflammatory disturbances present unquestionably greater variations from the typical than in any other organ; that is to say, the effects on the tissue in which the inflammation takes place are exceptional, on account of the peculiar character, physiological function and importance of the tissue, and unlike those of inflammation in other tissues. Nevertheless, we must admit the existence of similar anatomical lesions.

II. *Encephalomalacia in its narrower sense—that is, the softenings in the brains of the aged, and of those affected with heart disease*—or, more accurately speaking, the softenings from arterial thrombosis, from the embolic plugging of a cerebral vessel—must be ascribed to the formation of a hemorrhagic infarction, through plugging of a vessel. The conditions and genesis of this process have been accurately determined by means of a series of beautiful experiments (Cohnheim). The initial lesions have no connection with inflammation. Microscopically, too, they do not in any respect present the lesions of inflammation, but exhibit those which are now known to denote necrotic processes in the central nervous system. Except under very special conditions, a simple necrosis never leads to the formation of pus.

III. But as soon as a true encephalitis has ended—that is, as soon as those processes, which we must regard as really inflammatory, have run their course—the manifold processes of involution and resorption, by means of which the organism attempts to repair the injury already done, take possession of the affected spot. In this way it happens that, in a certain number of cases, the encephalitic focus melts away, and a cavity is formed, *the differentiation of which, from a thrombotic, embolic, or apoplectic cyst, presents the greatest difficulties, and is impossible if only the anatomical character of the cavity itself be taken into consideration;* the lesions in other parts of the brain, and in the other organs of the body, must be called to our aid in determining this question. This difficulty, to be sure, is not encountered in every case, but it has essentially contributed to the maintenance of the obscurity in which the subject has for so long a time been enshrouded.

IV. The circumscribed, punctated hemorrhage (infarct), which *leads to yellow necrotic softening*, and which, as has already been said, is, *ab initio*, absolutely unconnected with inflammatory changes, acts as an irritant to the surrounding brain tissue. Hence it is frequently surrounded by an *encephalitic zone*, by a "reactive," truly inflammatory process, which, however, does not attain the importance of an independent encephalitis in extent and acuteness. The inflammation is rather an accessory or secondary process, and, consequently, the initial lesion in the blood-vessel cannot be held directly accountable for it. It is unquestionably true that an encephalitis may accompany thrombosis and embolism, and hence the older observers cannot be reproached with a complete misconception of the process.

V. *Conversely, a partial necrosis of the surrounding cerebral tissue very frequently accompanies a true encephalitis.* Macroscopically it presents the appearance of simple yellow softening, and microscopically also it exhibits distinctly the characteristic signs of this process. It is not necessary to assume here a combination of inflammatory lesions with the above-mentioned changes in the vessels, for every partial compression which opposes an insurmountable obstacle to the entrance of blood into a small portion of the brain leads to necrosis of this portion. This was another source of the difficulties which for so long a time prevented the accurate differentiation of the different lesions.

VI. There are cases in which we may assume a combination of both changes in the central nervous system. *There are emboli of a specific nature, which possess a special tendency to excite inflammation.* These exert, in the places where they lodge, both a mechanical and a dynamic action. The immediate effect is the formation of a circumscribed, punctated hemorrhage; the specific irritation, however, which the embolus carries with it, immediately causes, at the affected spot, an intense inflammatory hyperæmia, an inflammatory exudation, and, in most cases, a rapid and active suppuration.

VII. *Finally, true encephalitic processes occur in the vicinity of neoplasms and of other collections in the brain which exercise pressure.* Here the combination of inflammation with yellow necrosis is very common. In most cases we are still unable to say whether the yellow softening is to be regarded as the termination of the encephalitis—that is, whether the simple necrosed spots were, at an earlier stage, in the condition of red softening, or whether their nutrition was directly impaired by growing neoplasm. However this may be, we have here again a combination of both conditions which is not difficult to comprehend.

VIII. It follows, then, that foci of softening (this expression we use in its widest sense) are met with in the brain, from the examination of which alone we can no longer ascertain the nature of the preceding processes. The peculiar combination in which inflammation and necrosis are very frequently found enables us to understand the long existing confusion of the two conditions; in the fresh state a careful microscopic examination will at once remove all doubt. We must always bear in mind that the cerebral tissue is, of all the tissues of the body, the one least capable of withstanding compression, and the one which most rapidly loses its normal texture when its functions cease; the combination of encephalitis with the manifold conditions of simple necrosis will then excite no astonishment.

General survey of the anatomical appearances.—We will disregard entirely for the present the varying size, position, and genesis of the foci, and confine ourselves to a general description of the processes so far as they are accurately known. It is to be premised that we have to deal always and under all circumstances with foci (Herde), that an acute inflammation of the brain in toto has never been seen.

The initial change in a portion of the brain which is attacked by inflammation consists in a striking change in its color. In a spot in which a reddish discoloration can already be recognized, a number of extravasations of blood, each about the size of a pin's head, appear, which are sometimes separated by bridges of tissue of different breadth, and are sometimes so close together that the existence of a confluent extravasation may be suspected. When the capillary apoplexies are numerous, the macroscopic appearances can scarcely be distinguished from those of a hemorrhagic infarct. Some encephalitic foci, however, present few points of extravasations, and there can be no doubt that the degree of the initial congestion determines the number and the closeness of the apoplexies.

In consequence of this initial change, the cause of which is doubtless to be found in the very marked softness of the neuroglia surrounding the vessels, the affected portion of the brain becomes more voluminous; it swells out, and the cut surface rises above the level of the surrounding parts. This peculiarity, however, is by no means characteristic of encephalitis alone; many hemorrhagic infarctions are also attended by swelling, and for a time at least exert pressure on the surrounding tissue.

The boundaries of the recent forms of inflammation are from the very commencement imperfectly defined. The surrounding tissues present different shades of color, in consequence of the imbibition of the coloring matter of the blood, which is set free in the apoplectic effusion. When the focus presents a deep red color, a red band forms its first boundary, which shades off into a brown, then into a yellow color, and finally into the normal color of the cerebral tissue.

The question presents itself here whether all cases of encephalitis commence in this manner. We will presently show that this question must be answered in the negative; there are abscesses of the brain which start as such from the very beginning and have not been preceded by a stage of red softening. The apoplectic extravasations into the inflamed part are rapidly followed by a great humectation both of the part itself and of the tissue surrounding it; in the latter this moistening takes the form of a more or less intense cerebral œdema, which may extend to a great distance, and may also present small capillary hemorrhages at points remote from the focus of disease. We regard this œdema as of inflammatory origin in one sense, although we do not believe that the transuded fluid is directly the product of inflammation. The universally increased cerebral pressure consequent on the swelling, which is often great, of a part of the brain, must certainly be capable of obstructing to a marked extent the circulation; as soon, however, as a retardation of the circulation sets in, the requisite conditions for a serous transudation are given. This question has assumed an entirely new aspect since Cramer found by means of direct measurement that the pressure in the jugularis sinks when the intracranial pressure is increased (injection of masses of wax between the dura mater and the bone). We shall come upon the œdematous transudations again in connection with abscess of the brain; they are there, indeed, occasionally causes of death.

In the diseased part itself, this humectation produces a macroscopically distinct loosening of the texture, which may be mistaken for softening. Since in most cases a great deal of blood is also extravasated, the designation red encephalitis is appropriate.

In pure encephalitis we have not met with a coagulating exudation. Those authors who speak of exudations appear to have assumed their existence for the sake of a theory. Rokitansky, in his admirable description of encephalitis, says that the exudation, when it possesses but little power of coagulation, permeates the focus equally in every direction, and makes the differences of color less apparent; but when it contains a considerable quantity of coagulable matters, these latter accompany the vessels in the form of irregular masses of greater or less size, or yellow and greenish striæ. This description is true to nature, but still we have always found in these yellow striæ either a commencing suppuration, or necrosis of the sheaths of the vessels. We will presently see that it is principally in these two directions that the further development of the circumscribed inflammation of the brain takes place.

The microscopic examination of the diseased spot at this stage reveals a number of changes, which we will group in three physiologically different sets.

1. *The changes which are due to the inflammation.*

In this connection, we must mention first of all the great hyperæmia and dilatation of all the vessels and capillaries, which is easily demonstrated. Hayem claims to have seen the vessels dilated to six times their usual calibre, but we have never met with such excessive dilatation. Further, the whole brain is dotted with groups and heaps of red blood-corpuscles, which, according to the length of time the affection has existed, present different stages of the change to lumps of protoplasm. That this change must exert a destructive influence on the neuroglia and nervous elements is evident; the effects of this action will be discussed directly.

The everywhere easily demonstrated *migration of the white blood-corpuscles* must, however, be regarded as the most distinct manifestation of the inflammatory disturbance. Rindfleisch finds pus first around the vessels which have bled. When cut across they are seen to be surrounded by a round border of white blood-corpuscles, which has pressed back the extravasated red blood-corpucles from the vessels from which they originated. These appearances have led Rindfleisch to accept, with some reserve, however, the theory that the pus is derived from the vessels. It must be admitted that the question whether the pus cannot be produced by division of the neuroglia nuclei, or by the proliferation of the cellular elements of the walls of the vessels, is still open to discussion. It has even been directly demonstrated by Meynert, that in the vicinity of encephalitic foci, especially in the œdematous border-zone, a division and a proliferation of the neuroglia nuclei take place. We have seen appearances which establish the emigration as an incontestable and certain fact, and we regard it as the chief source of the suppuration.

Suppuration is present under all circumstances, whatever be the subsequent fate of the focus of inflammation, but very frequently it requires a microscopic examination for its demonstration. The suppuration is an exceedingly variable element throughout the entire process, and only a small number of the encephalitic foci are transformed into macroscopic collections of pus.

2. *The changes which are due to the mechanical effects of the inflammatory disturbances in the tissue.*—The conditions of the mother soil in which the inflammation is located are in the brain entirely exceptional. There can be no doubt that the cerebral tissue is, of all the tissues of the body, not only the one in which destructive processes are most promptly excited by nutritive disturbances, but also the one which is most easily injured mechanically by pressure or displacement. It should, therefore, not surprise us to find throughout the whole region of the inflammation a great number of tissue elements (ganglion cells, neuroglia nuclei, nuclei from the capillary vessels, connective tissue elements of the sheaths of the vessels, etc.), which, having been separated mechanically from their positions, pushed aside, compressed, have been deprived of their normal nutrition. We are forced to accept the view that all these elements, in consequence of their physiological death, are changed into granule cells, and that this tissue change is the expression of an abolished nutrition. This can, however, be demonstrated much better in pure necrosis of the brain than in encephalitic foci, where a mechanical detachment of the elements from their original connections must be assumed.

This view is, to a certain extent, opposed to the views held by other authors. We fully acknowledge the correctness of the observation, that in encephalitic foci the cellular elements of the neuroglia and of the vessels are in a condition which Hayem claims to be identical with the cloudy swelling of Virchow, and which he is inclined to ascribe to an excessive imbibition of nutritive material. For us, however, this swelling and cloudiness in point of fact furnish no proof of an abnormally increased nutrition; but, on the contrary, they are evidences of a beginning decomposition from a perverted nutrition. We have been thoroughly convinced of this by the study of pure necrosis of the brain.

Consequently, we can ascribe the production of the great numbers of granule cells to this cloudy swelling. It is actually possible to follow step by step the transformation of the neuroglia cells, of the connective tissue cells of the walls of the vessels, of the nuclei of muscular fibres, and, finally, even of the ganglion cells, into the granule cells which are always found in great numbers in the foci. The endothelial cells are also transformed into similar elements; for when there are so many ruptures of the vessels and apoplexies of inflammatory origin, it cannot but happen that some capillaries will undergo necrosis. Especially instructive are the appearances presented by some small vessel of the brain, just large enough to possess an adventitia, which is covered with granule cells in such a way that it is actually enclosed in a sheath of these cells. For the rest we must regard the granule cells as final products which have no further function to perform.

The œdematous border-zone also presents changes in the elements which can scarcely be regarded otherwise than as the optical expression of severe disturbances of nutrition. Meynert has pointed out numerous changes of the ganglion cells in this locality, and any one who has ever examined the cortex of the brain in a case of acute diffuse encephalitis (dementia paralytica running a rapid course, and attended by maniacal excitement) will recognize some of these changes. The conclusions of Meynert, with regard to the changes in the ganglion cells, have been exceedingly valuable, and have, although they are perhaps not entirely correct in every particular, opened a new perspective for many views. The swelling up of the ganglion cells and the final molecular disintegration of the protoplasm, which Meynert describes, are indisputable and form a parallel to the analogous processes in the connective tissue elements. The vesicular transformation of the nucleus is doubtless an illusion; we have never seen a division of the nuclei in such localities; it is possible that we have to deal with a migration of white blood-corpuscles into the protoplasm. A sclerosis of the ganglion cells (Meynert) is frequently observed after the inflammatory processes have ceased, and we may also mention the calcification and pigment infiltration described by Foerster.

3. The third set of conclusions which is obtained from microscopic investigation of the foci of inflammation is rather of a negative character. *The nervous elements take no part in the inflammation*, the changes they undergo being due to mechanical destruction and necrotic processes. In this connection we accept the description given by Rindfleisch, because it corresponds most rigidly with the appearances. "The nerve-fibres within the circumference of the inflammatory focus are in part suspended in isolated fragments in the pus, in part still attached to the wall of the focus in a condition of progressing maceration and disintegration. I could discover neither fatty nor granular degeneration of them; drops of the medullary substance separate themselves from their surfaces; the axis cylinders gradually become more delicate and finally break up. The ganglion cells in the focus become dark, granular, and undergo destruction; I have been able to find well defined fragments of them" (Rindfleisch).

We have yet to mention a change of the axis cylinders which occurs with remarkable frequency: they do not isolate themselves in their medullary sheath, but simply become finely granular; then they acquire knobbed, globular swellings which, according to Roth, are hypertrophic nerve-fibres. The appearance is met with everywhere where the nerve-fibres are separated from their normal connections and are undergoing maceration. We must agree with Rindfleisch when he attributes the change to a maceration and swelling which precedes disintegration.

From these data, which we believe to be correct, it follows that even in the most recent encephalitic foci there is a concurrence of different processes, of a true inflammation with manifold degenerative processes, which are also met with under other circumstances where there can be no question of inflammation. Is it surprising, then, that the earlier investigators could find no dividing line between inflammation and necrotic softening, especially when we bear in mind that true necrotic softening, originating in vascular disturbances, may be surrounded by an encephalitic zone which is calculated to complete the similitude of the two processes?

Further Transformations of the Primary Focus.

With regard to the following changes, everything depends on the extent and situation of the inflammation, for a large proportion of the cases of encephalitis cannot undergo any further development, for the reason that life is rapidly destroyed. In a small proportion of the cases the inflammatory process ceases before a macroscopic infiltration of pus is produced; the process comes to a stand-still at a stage in which it presents the strongest resemblance to a primary necrosis with subsequent peripheral encephalitis. Hence some of the resulting conditions, also, are similar in the two affections, although they are essentially different in nature:

1. There is no doubt that an encephalitis of slight intensity and small extent may undergo an almost complete restitutio ad integrum. In consequence of the injury and destruction of isolated, though perhaps very small, portions of the brain tissue which always occurs, a perfect restitution is impossible. Small foci, however—for instance, the foci due to traumatic contusion (see etiology)—may disappear almost completely.

2. After cessation of the inflammatory process in larger foci, however, a residuum is left behind which contains the elements mentioned above. This undergoes the well-known destructive and reabsorbing changes. By degrees nearly all the cellular elements in the focus are transformed into granule cells, which undergo a gradual disintegration, and begin to form in the focus a thick emulsion, colored brownish or yellowish by the blood-pigment; in this way all the nuclei of the vessels and the neuroglia which are enclosed in the focus disappear, and its contents become more homogeneous. The walls of the focus are cleared in a similar manner by the disintegration of the débris; and, finally, at a certain stage of the process we have before us a focus of yellow softening, which gradually becomes more colorless, and at last may be transformed into a cavity filled with a

thin emulsive fluid. At this time all the signs of encephalitis in the focus itself have disappeared, and the diagnosis of the precedent inflammation can only be made from the concomitant lesions and from our knowledge of the course of the affection. What has become of the effused white blood-corpuscles during this time? This question cannot be answered with positiveness, but a great part of them, at all events, are changed into granule cells. We will have occasion presently to examine the yellow softening from another standpoint.

3. The acute observations of Durand-Fardel and Rokitansky, and many others, however, prove that the formative processes do not always cease entirely in such a focus; it is still capable of undergoing further development. We find, then, in place of the focus a fan-like, whitish, delicate stroma or texture supplied with delicate vessels, the interspaces of which are filled with a thin emulsive fluid. The stroma consists of very delicate connective tissue supplied with vessels; the formed elements of the fluid consist almost wholly of large quantities of granular fat and unknown albuminous bodies, together with a little free pigment. This fluid gradually becomes clearer, the spaces generally become somewhat smaller, and, finally, a chasm remains which is traversed by a number of delicate bands of connective tissue resembling septa, and is surrounded by somewhat condensed cerebral substance. We do not venture to decide from which tissue elements this new connective tissue is developed; that is a question on which we have had no experience. We will see later on, however, that the migrated white blood-corpuscles are destined, under certain circumstances, to take an active part in it. According to Durand-Fardel and Rokitansky, this peculiar transformation never takes place in the purely gray substance as, *e.g.*, in the cortex, but is met with especially in the white substance, and in those parts of the gray matter which exceptionally contain a large quantity of medullary substance. Rokitansky states that the focus last described is capable of an entire destruction. This cannot be contradicted, although all the traces of a resistant cicatrix could hardly disappear completely; he explicitly calls attention to the facts that apoplexy and infarct may terminate in the same way, and

that from the study of the lesions in the later stages the nature of the preceding affection cannot be positively determined.

4. Transformation into sclerotic induration. We do not here refer to multiple sclerosis of the brain, which is an entirely different affection from the encephalitis under discussion, entirely different in its etiology and in its course, and possessing different anatomical peculiarities.

Genuine local encephalitis may lead to the production of firm sclerotic cicatrices, which are located usually near the surface of the brain, more rarely deep in the interior of the organ (Hasse). These cicatrices are usually of a dirty-white color, tough, firm, and cut like caoutchouc or leather; there is a noticeable atrophy of the tissue surrounding them, so that the affected hemisphere is somewhat less voluminous than the other. Even distant portions of the brain—for example, extensive portions of the cortex—may be found in a state of atrophy, which doubtless dates from the very beginning of the process; we have already discussed Meynert's theory as to the manner in which the gray substance in the neighborhood of encephalitic foci is destroyed (œdematous peripheral zone). When these cicatrices are situated deep in the brain we can usually demonstrate the existence, at an earlier period, of cavities within them; for, as Hasse asserts, and as we can corroborate, they not unfrequently contain a nucleus which is of a different color from the rest of the cicatrix, and contains the last remains of the encephalitic focus: fat and pigment granules, hæmatoidin crystals, fragmentary and amorphous detritus. Such a cicatrix in the brain will naturally produce some impairment of the integrity of the cerebral functions; since it does not constitute a cure, a number of the symptoms of the acute stage will be continued into this chronic condition, or even new additional symptoms, both of a chronic and an acute nature, may be developed. It seems that such inflammatory processes in the brain very seldom become quiescent; they are followed by a gradually progressing atrophy of the entire brain which causes symptoms intra vitam, that even at the present day are frequently included among those of dementia paralytica. Hasse has described these peculiarities in the course of the affection, and this very experienced author has also called attention (p. 493) to the fact that an encephalitic cicatrix may at a later period excite a fresh inflammation, resulting in the development of a new girdle of red softening with capillary apoplexy. We may add that it may also excite a rapidly progressive, yellow softening, and that the latter may prove fatal. This point will again come under discussion in connection with the etiology.

Further, we have seen several cases in which the inflammation excited around embolic foci led to the formation of sclerotic capsules, several lines in thickness, which, being excessively poor in vessels, obstructed in a very great degree the process of resorption in the focus. The capsules consisted of an extremely dense, fibrous connective tissue which contained a large number of spindle cells with oblong nuclei. In one relatively recent case the examination convinced us that these spindle cells spring from migrated blood-corpuscles in the border-zone; there must consequently be some modification of the nutritive conditions in reactive encephalitis which permits the development of the latent powers of the white blood-corpuscles, while under other circumstances they undergo a physiological death.

It follows from the foregoing that encephalitis may lead to manifold subsequent conditions, about which we will add some observations when describing the symptoms.

5. Transformation into a collection of pus. First of all, we must discriminate between recent and old abscesses of the brain.

a. The recent cerebral abscess. In general, an abscess is said to be recent, or fresh, when it has been developed rapidly, and does not possess an enveloping capsule. It is evident that this definition is very indefinite. In the first place, we possess no sure criterion of the length of time that must elapse before an abscess can be called chronic; and, in the second place, a limiting membrane is often found on one side of the abscess, while on the other it is entirely wanting, and the contents of the abscess are in immediate contact with the cerebral tissue. There can be no doubt that abscesses of the brain which possess no limiting membrane have a tendency to acquire one. On the other hand, however, there are abscesses of the brain, provided with investing membranes, which have never gone through the stage of red softening or of the recent abscess, but which from the very commencement existed as encapsulated abscesses. We are, it is true, for the present unable to formulate a special category for these last abscesses—that is, we are unable to determine under what special circumstances and what etiological influences they are formed ; but we believe that the observation of a number of striking facts justifies the above statement. Consequently, the recent abscess of the brain would possess but a slight tendency to surround itself with a capsule.

In point of fact, an acute abscess possesses rather a tendency to spread in every direction, which is due especially to the character of the surrounding tissue. It presents an irregular cavity in the substance of the brain, whose walls have a rough, shaggy surface. The shaggy projections consist of portions of mortified cerebral tissue, which are attached to the larger blood-vessels. From the appearance of the tissue in the immediate vicinity, it is easy to see that complete mortification would have taken place in a very short time. We find the above described red softening, genuine inflammation of the tissue, involving to a variable depth the tissues around the abscess, and, in addition to it, many places where the color of the softened tissue is predominantly yellow, and where only the elementary processes of necrosis can be recognized. At a greater distance from the focus, the cerebral substance is œdematous ; for the particulars of this change we may refer to the remarks already made. It happens not very rarely that, in consequence of the pressure exerted by the abscess, a rapidly progressing yellow softening involves the whole of the surrounding tissue ; this must be attributed to a total suspension of the normal circulation, and consequently of the nutrition. This alteration of the surrounding tissues presents the most favorable conditions for the advance of the abscess ; in fact, it often enlarges unchecked in every direction, till it attains astonishing dimensions, and finally causes death by general pressure on the brain, or by cerebral œdema. It follows from the above that the contents of a recent abscess of the brain do not consist of pus alone ; in addition to the pus-corpuscles, the fluid contains a large amount of granular detritus and much fat, and on the borders of the abscess large numbers of granule cells which have been set free by the necrosis of the surrounding cerebral tissue. In the zone of the red inflammation there is a combination of the inflammatory with the necrotic processes, as has been already mentioned, while in the foci of simple yellow softening only the necrotic processes are found.

The enlargement of the recent abscess is brought about by the continuous melting down of its walls. There is an unquestionable connection between this and the continuous production of pus; but we do not yet positively know whence the large quantity of pus contained in the abscess is derived, and what elementary constituents of the brain are particularly active in its production.

We are compelled to assume that the greater part of the pus contained in an abscess of the brain is derived from the vessels of the neighborhood. We find also in the border-zone of a cerebral abscess the same circumvallation of the vessels by a ring of white blood-corpuscles which we meet with in a focus of red encephalitis which is undergoing the process of softening; further, we see that a number of those vessels from which the white corpuscles have escaped immediately undergo nutritive disturbances, which soon leads to necrosis. If this process take place throughout the entire circumference of the abscess, it is not difficult to see that a necrosis of that portion of the tissue which has escaped the mechanical injury of the extravasation, but which is now deprived of its nutrition, must follow. Consequently, the two processes, migration—that is, pus formation—and necrosis of the wall, would advance *pari passû.*

By virtue of its tendency to enlarge, the abscess now and then reaches the surface of the brain. As soon as the most minute perforation takes place, and perhaps even before, a most acute purulent inflammation of the pia is excited. This form of meningitis has already been mentioned. A perforation of the abscess inward is exceedingly dangerous. Perforations into the ventricles are relatively not very infrequent.

b. Old abscess of the brain, encysted abscess. It possesses, to distinguish it from the form just described, a fibrous capsule which may attain a thickness of several millimetres. This capsule is either in direct contact with the surrounding cerebral tissue, or different pathological conditions are interposed between it and the normal cerebral tissue. The histological relations of this investing membrane have been studied by Rindfleisch. Many points have been cleared up by the anatomical investigation ; but some very essential questions as to the origin, and particularly as to the development of the abscess, are still unsolved.

Rindfleisch affirms that there is a direct transition from the capsule to the surrounding nervous tissue. This connection—Hasse also calls attention to this point—in some cases is not very close, so that the abscess can be enucleated without much difficulty. The tissue in its immediate neighborhood may undergo a great relaxation, as Rindfleisch has demonstrated histologically. The histological structure of the capsule, according to Rindfleisch, is as follows :

The internal surface of the limiting membrane is smooth, and a continuous layer of cells in a state of fatty degeneration gives it an opaque, yellowish-white appearance. Outside of this there is a layer of regular germ-tissue, which has an unequal thickness, and, as a result, causes elevations of the surface.

Externally, this germ-tissue merges into a loose, distinctly stratified, spindle-celled tissue. Very perfect spindle-cells are found throughout the whole of this layer ; every apparently simple fibre, when carefully examined, can be demonstrated to be a spindle-cell, and from this it seems probable that the true connective-tissue fibres of the next outer layer are developed from spindle-cells. Now follows the true fibrous layer of the connective-tissue capsule, which, besides the fibres, still contains a large number of round and stellate cells. The latter become more numerous toward the external surface of the layer, where they assume for the most part the character of granule-cells.

Then follows another zone of fatty degeneration, which separates the capsule from the nerve-tissue. Rindfleisch believes—and he is undoubtedly right—that the pressure of the abscess prevents the normal distention of the vessels of the surrounding tissue, in consequence of which its cells undergo fatty degeneration. This degeneration, however, is not complete, for the softened zone is traversed by a network of coarse bands which contain primitive nerve-fibres, and, when the gray substance is the part involved, ganglion-cells; they are, however, in a compressed condition, and lie parallel to the surface of the abscess, so that the influence of the pressure of the abscess on them is apparent. The granule-cells of this softened layer are derived from the cellular elements of the neuroglia, which, according to Rindfleisch, undergo a process of division.

The pus of the abscess itself (Rindfleisch) is of a greenish-yellow color, of a synovia-like, greasy consistency, usually has an acid reaction (the reaction of the pus in most abscesses of the brain is acid), and is in the majority of cases odorless. The pus-corpuscles are mostly furnished with several nuclei, perhaps in consequence of the prolonged maceration in a slightly acid fluid. The modifications the contents undergo will be described below.

Unfortunately, all these data do not enable us to deduce satisfactory conclusions concerning the origin, and especially the subsequent growth of the abscess, which unquestionably takes place. Like many others, we, too, are unfortunately obliged to confess our inability to arrive at any positive decision as to the origin of the pus. With regard also to the origin of the capsule, the most competent judges will express only very guarded opinions. Rindfleisch regards the entire capsule, with the granule-cells of the zone of softening that surrounds it, as a production of the neuroglia, and his view has not yet been confuted. Since, however, it is evident that the pus in the abscess at certain times increases in quantity and exercises a pressure upon the surrounding cerebral tissue, which always leads finally to a fatal result, the question as to the origin of the pus must be considered the more important. The older hypotheses are inadequate to explain it. Lebert maintained that the pus was secreted by the smooth internal surface of the capsule, which resembles a mucous membrane. That the pus must be furnished by the investing membrane is evident, for we have no grounds to assume an independent cell-proliferation in the cavity of an abscess. Here, however, the possibility of exact statements ceases. We have ourselves endeavored particularly to demonstrate the occurrence of a migration from the vessels of the capsule; but our investigations have been entirely unsuccessful; they have not even thrown any light upon the processes in the germinal layer.

Whatever may be the *modus operandi*, after a very variable period of rest the abscess enlarges, causes manifold changes in the surrounding parts, and may reach free surfaces.

This enlargement exerts a destructive action on the surrounding cerebral substance. First of all, the intracranial pressure is thereby increased, and, as a consequence, the circulation in the brain is disturbed (retarded) to a variable extent. A number of the general symptoms that accompany an enlarging abscess must be attributed to this circulatory disturbance.

Moreover, the vessels in the immediate neighborhood of the abscess are compressed in a still greater degree, and this may lead to a rapid enlargement of the border-zone of fatty degeneration, so that yellow softening may be very extensive.

At the same time local inflammatory processes in more distant parts are met with; this is evident even from Schott's statements. This observer found the vessels in the neighborhood of the abscess distended with blood (encapsulated abscess of two and a half months' growth, with a soft pseudo-membrane), and their walls infiltrated with small, round bodies resembling pus-cells, so that in many vessels the walls were from five to ten times thicker than they should be. Schott attributes the formation of the capsule to this brood of young cells. What he saw was undoubtedly a migration of white blood-corpuscles. Hasse's hypothesis, that perhaps the induration of the circumference precedes the formation of pus, cannot be positively rejected in every case, because, as has been already stated, a limiting membrane may surround foci of true softening without suppuration. The hypothesis, however, cannot apply to every case.

Accessory changes in the brain: The perforations of the surface of the brain and into the ventricles have already been mentioned; they occur as well in connection with recent as with encapsulated abscesses. Abscesses situated in the middle of the parietal and frontal lobes open by preference into the ventricles. Here again, observers mention an acute suppuration of the ependyma of the ventricle, but we were unable to demonstrate it in a case in point. Abscesses of the middle lobe may open upon the base and give rise to diffuse acute meningitis of the base.

These are special complications, and the conditions that lead to them in individual cases are unknown. Other changes in the brain, caused by the abscess, are more important.

a. Widespread acute œdema of the brain.—This seems to have formed the only cause of death in a number of cases; and in fact, in some cases, it is exceedingly intense and widespread. It is hardly possible to account for it in any other way than by ascribing it to the retardation of the circulation produced by an intracranial pressure of moderate intensity.

b. Anæmia of the brain.—We do not believe this to be a direct cause of death, but regard it as an anatomical factum which has a great influence in the production of symptoms. When the tension caused by the abscess is great, the cortex is compressed against the inner surface of the skull, the convolutions are flattened and broadened, and the same appearances are produced as when there is great hydrocephalus.

c. Hydrocephalus internus chronicus.—Whenever an abscess is located in the cerebellum in such a position that it lessens the cavity of the fourth ventricle, or of the aquæductus Sylvii, a chronic exudation into the ventricles results.

d. More accidental lesions in the brain are very frequently met with; for example, injuries which will be described in detail when we come to consider the etiology.

e. There exists no direct connection between abscess and pachymeningitis; the conditions are more favorable for the production of the latter when the brain is in an atrophic condition (old abscess).

A spontaneous cure of an abscess of the brain has as yet never been demonstrated anatomically with absolute certainty; the possibility of a natural discharge can, however, not be denied. Several cases of cure after operation (trephining) are on record. Natural processes of cure have been observed in the abscess (Gull—calcareous transformation of the contents of an abscess), but no unquestionable instance of complete obliteration of an abscess of the brain has been recorded. The contents of abscesses have been evacuated in the following directions, after previous cementation of the periphery of the brain to neighboring parts:

Agglutination of the membranes; discharge of the pus through the point of adhesion and through the skull into the subcutaneous tissue (Bruns).

Perforation of the ethmoid bone and discharge of the pus into the frontal sinuses and nasal fossæ.

Perforation of the temporal bone; discharge of the pus in the neighborhood of the processus zygomaticus, under the temporal muscle (Wreden).

Perforation into the cavity of the tympanum, or adhesion of the surface of the brain and the petrous portion of the temporal bone, and secondary caries of the same (Odenius), an occurrence which is doubted by some authorities.

Perforation through the orbit, an occurrence of extreme rarity (Bauchet).

Subsequent extension of an abscess of the brain outward, and perforation through the old point of rupture (Gräulich).

We have given above a short description of the contents of an abscess of the brain. The statement that in diffuse and encapsulated abscesses different qualities of pus are found, by no means exhausts the subject. Many old abscesses contain pus which is much altered (fatty and broken-down pus-cells); frequently hæmatoidin is found in the pus, and once we found crystals of margarine and cholestearine; the assertion that corpora amylacea are found in it is somewhat doubtful. In certain cases the pus is exceedingly fetid (four out of eighty-nine cases, Hutchinson and Jackson; three out of sixteen cases, Gull; nineteen out of ninety cases, Rudolph Meyer.[1]) The latter, whose statistics we accept, classified these nineteen cases according to the etiology, as follows:

Otorrhœa	9	cases.
Fracture of the skull	3	"
Necrotic periostitis	1	"
Pyæmia	2	"
Chronic purulent inflammation of the lung	2	"
No cause given	2	"

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[1] Zur Pathologie des Hirnabscesses. Diss. Zürich, 1867.

The cause of the fetor of the pus cannot consequently be ascertained with certainty from these statistics alone; even if they contained a larger number of cases, the results would probably be no better. Affections of the neighboring bones, unhealthy suppuration, and an ichorous quality of pus, seem to be most frequently answerable for the peculiar quality of the abscess of the brain. The fetid abscesses are by no means uniform with regard to situation, position, or size; they may or may not be provided with a capsule.

Several authors have endeavored to discover the conditions on which the formation of the capsule depends—whether one distinct class of cases can be found which always possesses a capsule, and another which is always without it. *A priori* the answer of this question seems to be contained in the above; in general it is certainly true that acute abscesses do not possess a capsule, and *vice versa;* but still there are numerous exceptions to this rule. Abscesses that have existed for months and years have been found without investing membranes. Even the etiology and the mode of origin, as far as this can be ascertained, furnish no sure clews; traumatic abscesses, as well as those which are secondary to chronic affections of the bones, appear under both categories. Our own experience on this point confirms the already recognized rules, that the multiple abscesses of pyæmia never have capsules, and that the probability of encapsulation diminishes in proportion to the rapidity of the development of the abscess. We found the tension in encapsulated abscesses always greater than in those which did not possess a capsule; but a theory based on this peculiarity, that the greater tension of certain abscesses causes inflammatory reaction in the surrounding tissue and the development of a capsule, would undoubtedly be fallacious.

The question of the time when the abscess capsule is developed has been most carefully investigated by Rudolph Meyer. The following are some of the older statements:

Lebert collected eighteen cases in which the age of the abscess was ascertainable. In one case the demarcation was completed in eighteen days; in two cases in from twenty-two to twenty-four days; in four cases between the thirty-second and thirty-eighth days; in eight cases between the forty-second and sixtieth days; in three cases the time was still longer. Lebert believed the average duration to be from three to four weeks. Lallemand found, in one case in which death occurred thirteen days after the appearance of the first symptoms, the periphery of the abscess already covered with a soft vascular membrane; in another case, of from fifty to fifty-three days' duration, he found a membrane rich in vessels; in another, of thirty-seven days' duration, he found a "white, easily torn cyst-wall, resembling thickened pus." Schott believes that before the seventh or eighth week a firm cyst-wall is not met with. Gull thinks this limit is the shortest possible, and believes ten weeks are usually required. Finally, Rudolph Meyer found the following conditions: in a case of seven days' duration no trace of demarcation; in a case of thirteen days' duration an irregular, softened, slightly injected wall; in a case of eighteen days' duration the abscess did not possess a membrane; in cases of nineteen, twenty, twenty-five days, and in one of four weeks' duration, demarcation of the suppuration had taken place; in a case of six weeks' duration a resistant membrane, resembling a mucous membrane, was found; in older cases a capsule was present. He met with a few old abscesses without capsules.

We may add the following observations of our own to the above: abscess of thirteen days, without the least trace of a membrane; abscess of thirty-two days, no membrane; abscess of fifty-three days, with a delicate, membranous investment, containing distinct germ-tissue and a stratum of spindle-cells; abscess of eighty-three days, with a thick, firm capsule. This agrees with the statistics of Meyer, who places the appearance of a distinct capsule at about the seventh week. In determining this point he took into consideration only traumatic cases, the date of the beginning of which can be accurately determined.

The enormous size which abscesses of the brain sometimes attain is astonishing, and can as yet only be explained by theories which are not based on any positive anatomico-physiological facts. Abscesses have been described which involved not a mere lobe of the brain, but a whole hemisphere, so that only scanty remains of its tissue were still present. It is deeply to be regretted that accurate anatomical investigations have not been made into those cases in which, ex. gr., "the whole right hemisphere flowed out in the form of thick, greenish pus." The most striking point in these cases is the insignificance of the symptoms, especially the small number and slight intensity of the paralyses. It is an incontestable fact that paralyses may be absent in cases in which the destruction of a hemisphere is brought about slowly; we believe, however, from anatomical reasons, that, in such cases, at least a portion of the ganglia of the brain must be preserved. Actual proof of this has never yet been adduced, but neither has it been shown that the ganglia also were involved in the consumption.

On the other hand, many abscesses are exceedingly small. Pyæmic and embolic abscesses are occasionally only as large as a millet-seed; in other words, they are, from the very commencement, distinct abscesses. It is true that, in the later stages, inflammatory disturbances may be excited in the surrounding tissues, as has been already mentioned, but they are not preceded by a stage of red softening; the embolus appears, causes a small hemorrhagic infarct, and, in consequence of its infectious qualities, the resulting inflammation is so violent that it produces, from the very beginning, a purulent effusion.

The usual simple abscesses vary in size from a dove's to a goose's egg.

Abscess of the brain is either single or multiple, the multiple abscesses being less frequent; we counted twenty-nine instances of multiple abscesses in about one hundred and twenty available cases, a proportion which comes very near that found by Meyer (90 : 23, 80 : 22, Lebert). The multiplicity cannot be referred to any ascertainable causes. We may, it is true, suppose an infection of certain spots in the neighborhood of an abscess, through the lymph vessels of the brain, in consequence of which a number of abscesses are produced; but this has not yet been positively demonstrated. The embolic abscesses are usually multiple, and this accords better with current conceptions of the process.

Various statements are met with in the literature of the subject, concerning the predilection of cerebral abscess for certain parts of the brain. Hasse affirms that encephalitis occurs more frequently on the surface than in the interior, which is doubtless correct, for the reason that the great majority of the recent cases of encephalitis are due to injuries of the skull. The opinion of Gintrac—that the chief seat of abscess is the medullary substance, and not the cortex, and further, that the parietal and occipital lobes, and the cerebellum, possess an essentially greater predisposition to abscess than other parts of the brain—was long ago refuted. This opinion may possibly be correct, if the statistics include all varieties of cerebral abscesses. But if, as Meyer has done, we exclude all abscesses which are not dependent on local causes, we will find that no special predisposition is possessed by any part of the brain. If embolic suppuration in the brain were not so rare, it might perhaps be proved that it has a special predilection for the territory of certain arteries. A tabulation undertaken with this purpose, however, furnished no satisfactory results.

Etiology.

I. *Acute inflammation and recent abscess.*—In certain etiological particulars these two conditions cannot well be separated, and they will therefore be considered together.

A. The most frequent cause is *injury*. We must repeat in this place what was stated in connection with acute traumatic meningitis.

Inflammation of the brain has the same pathologico-anatomical basis as meningitis; both follow directly the different forms of contusion of the brain. It must be remembered, however, that the contusion may be peripheral as well as central; that it may be situated in the immediate neighborhood of the affected portion of skull, or on the opposite side of the brain, either peripherally or in the interior of the organ.

The primary traumatic encephalitis is most acute when the atmospheric air is allowed access to the wound, particularly to the deepest parts of it, to the pia and the surface of the brain. The inflammation of the brain is then coincident with the meningitis, and sets in within the limits of time which we sought to formulize for the period of incubation of acute meningitis. In superficially contused parts of the brain, under such circumstances, a process sets in which has hitherto not been mentioned. A genuine superficial mortification (Verjauchung)—a death, *in toto*, of the bruised parts—may occur, which does not, as in a simple encephalitic focus, merely involve single elements of the tissue, but fuses all the portions of the brain which have been deprived of their normal nutrition into a common gangrenous mass. The extent of this process depends on the degree of the contusion; if it be of slight intensity, the above described changes, peculiar to red softening, occur in the bruised part and its entire neighborhood. Sometimes, in the centre of the bruised part, a chocolate-colored, pulpy mass is found, which is surrounded by a broad zone of intense hyperæmia, with numerous capillary apoplexies. Next to this red areola comes the zone of inflammatory œdema—that is, an œdema consequent upon the inflammatory processes, in which small capillary apoplexies are also frequently found. The influence of this often widespread œdema upon the cerebral tissue must not be underestimated. If we bear in mind the already briefly mentioned discoveries of Meynert, with regard to the action of the œdema upon the elements of the brain, we will see that it may, in fact, be the direct cause of the interruption of the functions of the affected part.

The microscopic appearances have been previously described.

The concurrent meningitis, with its intense action upon the cortex far beyond the boundaries of the primary focus, in conjunction with the action of the encephalitic focus upon the brain, and particularly on its circulation, rapidly leads in very many cases to a fatal termination. As has been already mentioned, however, meningitis does not necessarily occur, and the encephalitic focus, then, has time to undergo further transformations. These are:

a. When the discharge of the secretion is unimpeded, a cure of the encephalitis is possible. When the lesion is not very intense, the brain, like other tissues, is able to produce granulations—a process of which the minute mechanism is still but very imperfectly understood.

These granulations blend with those which are furnished by the membranes of the brain and the injured bone, and the result is a cicatrization of the gap in the skull. At first the whole forms a tolerably tense cicatrix, which, within a certain circuit, binds together all the parts involved. Later, the brain appears to retract inward by virtue of the gradual stretching of the cicatrix; at least, we find between the superficial covering and the surface of the brain a loose connective tissue whose meshes are filled with a serous fluid. The brain, however, presents a depression on its surface which not unfrequently corresponds to a dilatation of the lateral ventricle.

b. Acute suppuration.—Acute superficial suppuration, which is not limited by a capsule, and presents the already mentioned characteristic peculiarities of the contents and of the surrounding tissues, is unfortunately a much more common termination than that mentioned under *a*. Meningitis is invariably present, and we must here repeat what has already been said—viz., that in the majority of cases of perforating wounds, with contusion of the brain, the post-mortem appearances are made up of a combination of these two conditions.

This acute suppuration is not confined to the limits of the superficial contusion; not very unfrequently a number of smaller abscesses are found grouped around the superficial one. In the majority of such cases it is impossible to determine whether we have to deal with foci of central contusion, or with a transmitted infection of the parts surrounding a superficial contusion. It is evident from the nature of the injury that the suppuration in this category of cases must be peripheral; abscesses of this variety are absolutely the most frequent of all cerebral abscesses.

The time which elapses before the development of an acute abscess is exceedingly variable. Beck found an abscess the size of a pigeon's egg in the frontal lobe of the brain on the fifth day after a gunshot wound. We have seen one such after thirteen days. According to Bergmann, however, the average duration is from three to five weeks.

c. Yellow softening.—A contused wound of the brain, which is followed by encephalitis, does not necessarily lead to suppuration. Lesser degrees of contusion, especially those which produce merely punctiform hemorrhages, provoke only a slight degree of inflammation, and in consequence of this, the elementary changes of the necrosis are so much more prominent than those of the inflammation, that after a short time the macroscopic appearances of yellow softening are produced. The microscopical appearances have been described above.

In a number of cases, however, further changes occur. The yellow softening has a peculiar tendency to extend, which *à priori* does not seem to be grounded in its nature, but which is intelligible when we bear in mind the characteristics of that œdematous border-zone, which is in reality already in a condition of quasi-necrosis; at all events, isolated changes take place in its elementary constituents, which cannot be otherwise interpreted. Hence, it is possible for the softening to extend, and under certain circumstances it may involve very extensive portions of the brain. The clinical history of this progressing yellow softening has not yet been accurately constructed; it is certain, however, that the last stage of yellow softening may resemble very closely that of chronic abscess of the brain. The last stage of abscess is by no means always characterized by very imposing symptoms, such as paralyses, convulsions, etc.; a simple coma sometimes ushers in the fatal termination in both affections. On the other hand, yellow softening is sometimes attended by convulsions confined to certain sets of muscles, which we are as yet unable to explain.

d. It seems that, under certain favorable conditions, a contusion of the brain may not be followed by encephalitis and yellow softening, or even by meningitis, although the skull-cap is opened. This can only be the case, however, when the contusion is very slight and circumscribed, and consists merely of a small number of capillary apoplexies. In this connection those post-mortem appearances are important which consist merely in a slight callous thickening around a small central nucleus, that contains fat granules and a few granule-cells and crystals of hæmatoidin. In the preceding paragraphs we have described the course of the encephalitic processes, which follow perforating wounds of the skull. When there is merely a fissure, in which no pressure is exerted on the brain, either by depressed fragments of bone, or by a foreign body, the effects of the contusion of the organ may be just the same. Meningitis and encephalitis may run their course with great rapidity, and other processes also occur; but here the affection often terminates in chronic abscess of the brain.

Finally, those contusions of the brain which occur without any perforating wound of the skull excite encephalitis just as well as those which are exposed to the atmospheric air. The inflammation in these cases very generally terminates in chronic abscess of the brain. This fact is very striking, for we have reason to suppose that a traumatic apoplexy does not differ very much from an ordinary one, which constitutes also a serious injury of the brain, and yet the latter never produces pus.

Acute inflammation of the brain, after the fontanelles have closed, follows anatomically the same course as when the fontanelles are still open, except that the necrotic processes remain more in the background. We seldom have an opportunity to study it, partly because the affection is not a common one, and partly because, before death occurs, the encephalitis has frequently already developed into a cerebral abscess.

But it is important to know that in the closed skull encephalitis does not *necessarily* follow. Several cases have come under our observation where contusion of the brain, even under unfavorable circumstances, only led to very circumscribed yellow softening; if inflammation existed at all, it must have been of a very trifling nature. Indeed, in one case a small splinter from the vitreous plate had perforated both dura and pia, and lacerated the surface of the brain; but there was not even a trace of inflammation; it produced simply a small spot of yellow softening, which extended to the white substance. What has been said above, however, applies also to such yellow softenings. They may also remain latent for a long time, and then, under conditions that are entirely unknown, a rapid extension of the softening may set in and lead to death.

Acute encephalitis has also been known to follow operations on the surface of the skull, even when performed with all caution. This undoubtedly constitutes the first stage of those cases which will be spoken of presently in connection with circumscribed abscess of the brain. In one case (extirpation of goitre) a focus of encephalitis as large as a nut was found in the frontal lobe, although there was no affection of the bone and no inflammation of the dura or pia. In a second case red encephalitis of the frontal lobe appeared five days after the extirpation of an epithelioma from the orbit. The mode in which the inflammation is transmitted to the interior of the brain is as yet wholly unknown.

B. *Affections of the bone in the vicinity of the brain.*—We are unable to state positively whether all cerebral foci occurring in connection with caries of the petrous portion of the temporal bone, and collections of pus in that bone, were in the commencement cases of red softening. It is asserted, on the one hand (Hasse), that caries of the skull produces principally recent abscesses, and that these always have a red prodromal stage; on the other hand, we have seen cases of caries of the petrous bone where several small cerebral abscesses, the size of millet-seeds, which presented distinct lines of demarcation from the adjoining healthy brain tissue, were situated beneath the surface of the cortex. Evidently different processes are met with—processes of great intensity, which induce rapid softening, as well as slower and entirely different infection of a portion of brain, which from the very beginning is attended by the production of pus.

C. *Various circumscribed affections of the brain.*—The occurrence of inflammatory softening of the brain around tumors, which may terminate in the formation of pus, is incontestable. The tumor is found to be surrounded by an encephalitic, and outside of this by an œdematous zone. Between the red areola and the tumor itself we not unfrequently find a zone of yellow softening, in which a necrosis of both the inflamed and the normal brain tissue has been produced by the pressure of the tumor. This then reacts on the tumor itself in a different way—relations which do not require description here. Reactive inflammation of the brain is also met with in connection with all forms of infarct and apoplexy. The very marked differences in the lesions produced by these processes cause similar differences in the intensity and extent of the encephalitic changes in the vicinity. We do not know of any instance in which the encephalitis in these cases terminated in suppuration; that it may lead to the production of a sclerotic capsule has been already stated. The subsequent increase of the primary loss of tissue is frequently due to this encephalitis.

D. *Acute diseases.*—Encephalitic foci are said to occur in a number of acute febrile affections. In this chaos of inaccurate accounts it is extremely difficult to form an accurate judgment. It is especially typhoid and its kindred diseases that have been said to give origin to localized red softening of the brain. The different published descriptions at least fail to convince us that true primary inflammations of the brain have ever occurred, if we except the multiple cerebral abscesses produced by metastasis from the lungs or other organs (Hofmann). We have, it is true, observed a number of circumscribed cerebral lesions in typhoid, but they exercised no essential influence on the course and termination of the disease; they were always simple necroses, without any encephalitic disturbance. Their etiology, it must be admitted, could not be ascertained; but it was certain that they were neither pyæmic nor septicæmic in nature. Nothing positive is known about the connection of petechial typhus with these cerebral lesions, although they sometimes occur together. In severe forms of intermittent fever cerebral foci have also been described (Titeca), but their characters were not explained. Scarlet fever presents encephalitis in the form of an acute suppuration in the brain; but here the brain has been infected by neighboring suppurative processes. The causes of the localized cerebral affections that complicate measles (Hannon, Rilliet, Mayo, Barbieri), and their true nature, are still uncertain. In variola there is a decided tendency to localized inflammatory processes in the brain, and Westphal has also found them in the spinal cord. Aphasia and paralyses, which can only be ascribed to inflammatory foci, are mentioned by Curschmann (*vide* his treatise in this work). One focus that came under our own observation in a case of variola was a simple necrosis. Our own investigations have shown that the circumscribed affections which are met with in cases of carbonic-oxide poisoning are necrotic in character, and have no connection with encephalitis.

We call particular attention to the fact that we have in the above referred solely to localized inflammation. We do not thereby deny the occurrence of diffuse affections of an inflammatory nature in all these diseases; on the contrary, we are among the most decided advocates of that view.

E. *Affections of the Heart.*—There appear to be a number of cases in which abscess of the brain is caused by a simple arterial embolus from the heart. The possibility of such an occurrence is beyond question; we have ourselves observed a case of ulcerative endocarditis where a multiple embolism of the brain had caused several small encephalitic abscesses about as large as beans, together with other small non-inflammatory infarcts. In comparison with the great frequency of acute and chronic endocardial processes, these appearances are exceedingly rare, and in point of fact occur only when the embolus has a special character (gangrenous destruction, mycosis).

F. Red encephalitic softening, as a complication of *suppurative and sloughing processes* in different parts of the body, is far more rarely seen than abscess of the brain. Perhaps it is true of this variety of abscess, too, that in a certain number of the cases the suppuration is not preceded by a macroscopically evident stage of red softening; both capsulated and recent abscesses have been found under such circumstances, and the above supposition applies only to the first. The primary infecting focus is in most cases situated in the lung, and, as we will learn later on, chronic putrid bronchitis and bronchiectasis, with their resulting conditions, are especially liable to act as causative agents; however, the number of these cases in which acute encephalitis has been found is exceedingly small. There can be no doubt that the same process can be excited in the brain by unhealthy suppurations in other parts of the body also, but in point of fact our knowledge of the primary process in the brain is limited to the inferences which can be drawn from the cerebral abscess.

G. Is there a spontaneous, circumscribed inflammation of the brain? In the literature of the subject we find very few cases which could be allowed to pass as such, for which no cause near or remote can be discovered, so that nothing remains but the supposition of a spontaneous inflammation of the brain. To writers on abscess of the brain, the existence of idiopathic abscess is becoming daily more problematical. We quote a case reported by Hayem, which, at the first glance, conveys the impression of a spontaneous encephalitis; at all events, the affection is exceedingly rare.

34. A hard-drinking man, fifty-eight years of age, fell senseless on the pavement, and lost forthwith the power of speech and of moving his left arm. After six weeks an incomplete paralysis of the left side still remained, and the mental capacity of the patient was very much reduced; he could not read nor write, and could speak but very few words; he soon sank into a state of somnolence and coma, and died. The convolutions on the right side were flattened; in the posterior white nucleus of the hemisphere, corresponding to the outer surface of the thalamus, as far back as the posterior end of the corpus striatum, was a swollen part, which was dark in the centre and of doughy consistency. The greater part of the affected spot was very vascular and still contained many well preserved nerve-fibres, together with some that were varicose and studded with shining granules. In the interstitial tissue there was a great number of elements resembling nuclei, many of which were crowded closely together; on the vessels there were fat granules.

In spite of the fact that the description accords with encephalitis, this case must, like many others, be accepted with a reservation; we do not believe that it demonstrates in a satisfactory manner the spontaneous occurrence of encephalitis.

II. *Capsulated abscess.*—The etiology is, for the most part, the same as in the recent abscess:

A. Injury again plays the principal rôle. Lebert found it to be the etiological factor in a sixth of his cases, Schott in thirteen cases out of forty, Meyer in twenty-one out of eighty-six, and our own statistics give exactly the proportion of one to four. We have sufficiently pointed out above the circumstances under which the abscess of the brain arises, and here we will only lay stress on the fact that capsulated abscesses are found with relative frequency in skulls that are completely closed, corresponding to the fact that circumscribed contusions of the brain occur also without the slightest injury to the skull (Gull, Sander, Wyss, Beck, and many others).

In this case, too, it is a fact that the abscess is by no means necessarily situated at the point where the injury was received; it may be situated on the opposite side of the brain (contusion by contre-coup). (Ziegler, Bruns, p. 986.) Not unfrequently splinters of bone, or foreign bodies, are found in the abscess at the autopsy; it has already been stated that splinters of bone may exist in the closed skull, without giving rise to suppuration.

The ultimate cause of suppuration in the brain is unfortunately unknown. That causes of a special nature must exist, which are present in some individuals and not in others, is evident from the fact that a number of contusions of the brain are not followed by suppuration; nevertheless, these cases manifest no appreciable differences from others which do lead to the formation of abscesses.

B. Diseases of the ear, caries of the bony portions of the external auditory canal, as well as caries of the walls of the tympanic cavity, of the walls of the labyrinth, and finally, of the mastoid process.

Abscesses of the brain, which are secondary to affections of the ear, appear to be slightly more numerous than those which arise from injury.

The cerebral abscesses are situated most frequently in the hemispheres, less frequently in the cerebellum, and, in very rare cases, in the pons. It is a very striking fact that the right hemisphere is much oftener the seat of abscess than the left (fourteen right, four left, Meyer; our own statistics give the same proportion). Multiple abscesses are very rare (Meyer); the great majority of them are unilocular. We find it very frequently stated that the pus contained in the abscess was ichorous and stinking.

There are apparently two ways in which the affection of the ear may lead to suppuration in the brain. Some abscesses are directly continuous with perforations of the carious bones, from which an infectious fluid oozes out, which excites a circumscribed inflammation of the membranes, terminating in perforation, and then comes in contact with the surface of the brain, where it evokes suppuration. The resulting abscess appears to be merely a superficial defect of the brain, and has the diseased pia and dura as a covering. A communication with the diseased bone is the result, and the patient will be very fortunate if he escape general purulent meningitis altogether, or even for a time. Or the perforation of the dura occurs under similar circumstances, and then an infectious tissue-element obtains access to the interior of the brain in some way as yet unknown, and excites suppuration there. The abscess may be surrounded, even towards the diseased petrous bone, by healthy brain tissue. The enlargement of the abscess, which soon takes places, causes in some cases a rupture of the attenuated outer wall, and perforation follows. The first variety of abscess would, therefore, correspond to a direct infection, the second to a metastatic transplantation of the suppuration.

The perforation of the bone is, however, not a *conditio sine qua non* for the existence of an abscess of the brain. Many cases are on record where the surface of the bone was perfectly intact; where, indeed, the existence of caries could not be positively demonstrated. We may refer here to the observations on osteophlebitis of the petrous bone, made while discussing the meningitis which is consecutive to aural diseases. It is true that, in the majority of cases, the consequence of the osteophlebitis is a thrombosis of the sinus (directly observed by Wendt); but we have had an opportunity to convince ourselves that a thrombus of the sinus extending upward, and in a state of purulent disintegration, was the cause of an abscess of the brain.

If the cerebral abscess is often of metastatic origin—which we doubt,—at all events the affection cannot be transmitted by metastasis to distant parts of the organ, for the abscesses are always found in the immediate neighborhood of the seat of the primary disease.

A number of modifications can now be easily made out:

The cerebral abscess most frequently follows a perforation ot the roof of the tympanic cavity; this perforation is due to caries of the wall of the middle ear, the mucous membrane of which has usually been for a long time in a condition of purulent inflammation. This abnormity is not necessarily the only change in the petrous bone. Usually the dura is raised by the pus, and very frequently it is necrosed; but beyond the affected spot it is more firmly attached to the still healthy parts of the bone. Frequently also it is adherent to the pia and the surface of the brain, and is converted into a protecting mass of callous connective tissue. The abscess is situated close by the temporal lobe, and is sometimes connected, by a fistulous canal, with the opening in the bone (Wendt), so that a communication exists between the abscess and the cavity of the tympanum. In this manner the otorrhœa may be actually cerebral. Wendt deduced from this fact a valuable therapeutical point, viz., that injections with strong pressure should, under all circumstances, be avoided.

The case is somewhat different when, instead of a perforation of the roof of the tympanic cavity, an opening takes place from the cells of the mastoid process into the middle fossa of the skull (Wendt). The abscess, in such a case, is also located in the temporal lobe, and its relation to the opening in the bone varies in accordance with what has already been said in general on this point.

It is more usually the case that the perforation from the mastoid cells takes place on its posterior wall; in such cases the cerebrum incurs but little risk, but the cerebellum is jeopardized in the highest degree. One of the best described cases is reported by Wendt (abscess in the right side of the cerebellum as large as a cherry, loose adhesions of the dura to the posterior surface of the pyramid, two perforations in the former, a perforation in the covering of the processus mastoideus five millimetres in diameter; the posterior surface of the pyramid in its entire extent rough and bathed in pus, transverse sinus imbedded in pus and detritus, thrombus of the same in a state of caseous degeneration, thrombosis of the vena jugularis interna).

Several times the perforation which had caused the abscess of the cerebellum was on the posterior wall of the cavity of the tympanum. Further cases have been described (Hutchinson) where the posterior wall of the external auditory canal presented a perforation consecutive to caries of the bone; the result was abscess of the cerebellum. Finally, numerous cases are on record (Schwartze) which prove that the abscess of the cerebellum is by no means necessarily preceded by perforation of the bone. A purulent catarrh of the middle ear may excite purulent inflammation of the soft parts of the labyrinth, and from here the suppuration may extend along the facialis and acusticus, and establish itself in the cerebellum. A few authors (Gull) assume that the inflammation can be transmitted, by means of phlebitis of the small veins and of the vena aquæducti cochleæ, to the cerebellum. Abscesses in the pons (Hirnstamm) are rare; the material is too small to enable us to form a correct judgment as to their mode of origin; usually thrombosis of the sinus is present. In a case reported by Wendt, along with thrombosis of the sinus and abscess in the pons, the roof of the cavity of the tympanum was carious, discolored, and presented several small perforations, and the dura was raised.

Finally, abscess of the brain occurs without any perforation of the bone, and without affection of the dura, in connection with a less widespread carious affection of the petrous bone; we have briefly indicated above the manner in which this can occur.

From these statements certain rules with regard to the implication of particular parts of the brain may be deduced which Toynbee reduced to a formula. According to him, the inflammation of each portion of the organ of hearing is transmitted to a particular region of the brain; thus the cavity of the tympanum stands in a relative connection with the cerebrum, the meatus auditorius externus with the sinus lateralis and the cerebellum, the labyrinth with the medulla oblongata.

This law of Toynbee has been modified by Gull, who assumed that the cerebellum and sinus lateralis are threatened in disease of the processus mastoideus, and the cerebellum alone in caries of the cavity of the tympanum. Meyer has in general confirmed this rule, but has added that caries of the posterior wall of the meatus auditorius externus, as well as caries of the processus mastoideus, can give rise to disease in the cerebellum. Finally, we must add the cases of abscess of the cerebellum without perforation, where the suppuration—as in meningitis—travels along the acusticus and facialis. These constitute an exception to the law of Toynbee and Gull.

It is evident from the above that thrombosis of a sinus constitutes frequently an important connecting link. Purulent meningitis also often accompanies or follows the above, so that really, in order to attain a complete understanding of the subject, all three affections should be described as parts of a process capable of undergoing different modifications.

Since diseases of the ear are very frequently caused by acute diseases (typhoid, measles, scarlatina, erysipelas, variola, etc.), these latter should be admitted as remote causes of abscess of the brain. It is a well-known fact that all forms of inflammation of the ears are specially frequent in scrofulous persons, and in these cases the exudation usually undergoes the caseous degeneration.

C. *Nose, antrum of Highmore, orbit.*—In a small number of cases the inflammation was transmitted to the brain from the nose and the antrum of Highmore.

Maas mentions abscess of the brain following the partial removal of a naso-pharyngeal polyp. Simon (Brit. Med. Journ., June, 1858) speaks of polypous growths in the nose and frontal sinus which had caused absorption of the frontal bone, inflammation of the dura, and an abscess in the anterior lobe of the brain. Gull mentions two more cases where the inflammation was transmitted from the nasal to the cerebral cavity. Main observed a case in which a periostitis of the root of a tooth in the upper jaw gave rise to an inflammation of the antrum of Highmore; a caries of the os ethmoideum developed from this, then an abscess in the back of the orbit which forced its way through the foramen opticum into the cavity of the skull, and excited a fatal arachnitis and encephalitis. We have observed a case which, however, belongs more properly in the following category, where a caries of the roof of the orbit with consecutive abscess of the brain was developed after a stab wound of the orbit above the eye.

D. *Caries of the bones of the skull, with the exception of the petrous bone.*—A few cases have already been cited; the reports furnished by Wendt, of several cases illustrating this cause, are very interesting; further, the cases observed by R. Meyer belong here.

E. *Suppuration around tumors* is much less frequent than red softening; but few instances of it have been recorded. Webber has described an abscess of the cerebellum which had developed around and in the substance of a glioma, but the case is not entirely convincing. Cases have also been reported by Lutz (abscess in the frontal and temporal lobes around a tumor located on the convexity of the left hemisphere), by Virchow (cholesteatoma of the petrous portion, with purulent arachnitis and an abscess in the cerebral tissue), one by Hasse, and one not very striking one, by ourselves (purulent arachnitis of the base, and purulent softening of one hemisphere of the cerebellum in connection with a sarcoma of the base).

F. *Metastatic* (*embolic*) *abscesses of the brain.*—Although the absolute proof of the direct embolic origin of an abscess of the brain, the accurate demonstration of the infectious embolus, is still wanting, we find nevertheless important evidence of it in an observation of Boettcher. He found in the cavity of an abscess of the brain, which was consecutive to an abscess of the lung, a pigment which he was able to declare to be lung-pigment. This observation is of great value; there can be no doubt that some broken-down tissue in the lung obtained access to the blood current, and was conveyed to the brain from the left side of the heart.

The lung is the organ from which the infection of the brain is most frequently derived. Probably in most of these cases we have to deal with broken-down thromboses of the veins of the lung. The affections of the lung which may cause metastatic abscess are:

1. Bronchiectasis with stagnant and putrid secretion (Gull, Biermer).
2. Bronchiectasis with consecutive gangrene of the lung (observed by ourselves).
3. Circumscribed gangrene of the lung in typhoid, multiple abscesses of the brain (Hofmann).
4. Abscess of the lungs (Boettcher).
5. Tuberculosis (Meyer, Biermer). Gangrenous tuberculous cavities (one case observed by ourselves; small cerebral abscess situated above the *nucleus lentiformis*, without any traces of red softening).

6. Putrid bronchitis with consecutive pneumonia and gangrene of the lung (Meyer).

7. Chronic pneumonia due to the presence of a piece of bone in a bronchus, abscess of the brain (Bamberger).

Usually, when the abscess is secondary to putrid affections of the lung, its pus possesses the same putrid character.

Abscess of the brain is much less frequently secondary to abdominal affections. But few accurately described instances are on record: abscess of the right ovary, liver, and pleura; finally, abscess of the brain (Biermer, reported by R. Meyer); old dysentery, abscess of the liver—abscess of the brain (observed by ourselves); resection of the head of the femur, multiple abscesses of the brain (Maas). We also find on record: an old abscess of the musculus rectus abdominis, abscess of the brain; suppurations of the mesenteric glands, purulent spondylitis, and also a number of incomplete observations of small value.

Acute pyæmia, after injuries of the periphery, produce in some cases multiple acute abscesses of the brain, which do not possess capsules. Surgical literature contains a considerable number of such cases. Physicians who have devoted themselves especially to the diseases of children, report cases of encephalitis and abscess of the brain which followed abscesses of the subcutaneous connective tissue, especially after vaccination (Bednar), inflammations of the joints, and caries of the bones.

G. *Idiopathic abscess of the brain.*—Although we have met with several cases which seem to prove that there are cerebral abscesses which do not possess any demonstrable external cause, we are very far from assenting to the doctrine of the so-called idiopathic abscess of the brain. It is instructive to see how all the more recent observers are gradually discarding this doctrine. Of Gull's seventeen cases, only one is classed as an idiopathic abscess; in R. Meyer's observations there is also only one such, and in that case the anamnesis was *entirely* wanting. A large number of so-called idiopathic cerebral abscesses which are contained in modern literature do not by any means present satisfactory guarantees of absence of the known etiological influences. In the one particular case observed by ourselves, which we were most inclined to consider idiopathic, the traumatic nature of the affection was subsequently discovered. We have, therefore, for our own part, been forced to the opinion that an idiopathic suppuration of the brain does not exist.

Abscess of the brain is more frequent in males, which evidently depends on the greater frequency of injuries in that sex. We find the greatest number of cases between the twentieth and thirtieth years of life, for the same reason. Even during the earliest period of childhood, abscesses of the brain have been found, but the number of such cases is small. (Wyss, eighteen cases from one to fifteen years of age—twelve boys, six girls.) After the sixtieth year cerebral abscesses are very rare; we have met with only one case, and in that the patient was sixty-seven years of age.

Symptomatology.

I. *Acute encephalitis.*—It is exceedingly difficult to say anything about the symptoms of acute encephalitis which will apply to all cases. A very great majority of the cases described under the name of encephalitis are entirely worthless for the symptomatology of that affection; they are in reality cases, not of encephalitis, but of necrosis of the brain-substance, which is developed more or less rapidly, to which a reactive encephalitis, often of very subordinate importance, is superadded. Those authors who have confined themselves to their own observations have invariably come to the conclusion that they have seen very few cases of genuine, recent, localized encephalitis. If we turn for help to surgical literature, we find that an accurate symptomatology of encephalitis cannot be gathered from it either. Bruns and others speak not without reason of a combined clinical history of meningitis and encephalitis, for it is very seldom possible to differentiate the two. The otorrhœic forms are seldom seen in the stage of red softening; probably this stage does not in all cases precede the abscess of the brain. The secondary encephalitis developed around tumors is by no means calculated to bring into view the pure symptoms of the uncomplicated affection; the encephalitis which accompanies suppuration in other parts of the body is very seldom seen in the primary stage of red softening. Finally, in our opinion a spontaneous localized encephalitis does not exist. In those cases which have been classed as spontaneous, the real causes have been obscure; moreover, these cases of apparently spontaneous encephalitis are exceedingly rare, so that no general conclusions can be deduced from them.

a. Traumatic form.—We are obliged to make use again of the clinical history of contusio cerebri which we have already described. We saw, then, that the different varieties of injuries of the skull were attended by very different symptoms, which at one time resembled those of commotion, at another those of compression, and that when the brain itself was injured a number of the symptoms of a local disturbance might be produced, from which, in some cases, the diagnosis of the part of the brain involved could at once be made. The meningitis which follows such lesions, with its chief causative agencies, have also been described above. While speaking of the morbid anatomy we pointed out that the injuries which led to meningitis are, in the great majority of the cases, superficial contusions of the brain, but that contusions can also take place in the interior of the organ. These two varieties of contusion are about equally liable to be followed by acute localized encephalitis, but the termination of the inflammation is apt to be different in the two cases. The superficial lesions lead to red softening with consecutive, recent, diffuse suppuration of the brain in the great majority of the cases, provided they be exposed to the air; the deep contusions may also be followed by red softening and suppuration; but in a number of the cases this has a tendency to keep within limits and to develop into chronic capsulated abscesses.

In both cases the stage of encephalitis without suppuration is relatively short and transitory, and its symptoms can hardly be isolated. When an injury to the head is followed by the general symptoms of concussion or of compression, the stage of acute inflammation, provided a contusion of the brain have occurred, may begin even during the period of unconsciousness, although this perhaps lasts only a short time. The patient perhaps regains consciousness, but in a short time is seized with the previously described symptoms of diffuse meningitis. This meningitis alone is diagnosed, and the physician is then surprised to find at the autopsy a peripheral spot of red softening which is in the process of transition into diffuse suppuration, or an already formed abscess. Hence, in surgical cases it is scarcely ever possible to differentiate with certainty peripheral, diffuse suppuration of the brain from diffuse meningitis, except when the peripheral contusion of the brain has involved one of those superficial parts which we know to possess distinct functions (frontal lobe, centres of Hitzig, the walls of the left fossa of Sylvius and its neighboring parts), when consequently the symptoms of a local affection, isolated paralysis, isolated convulsions, aphasia, are added to the symptoms of the diffuse meningitis (see preceding section on contusion of the brain). But even then the diagnosis of a peripheral encephalitis is by no means sure; for it is possible that we have to deal with an encephalitis located beneath the cortex, which has lacerated the motor nerves leading downward, or which has involved the cortex of the island (Insel) from within out. In these cases of injury death usually occurs in the second week, or at the commencement of the third. No universally applicable rule can be deduced from the duration of the disease. It has been said that a rapid course (about one week) with diffuse symptoms indicates a wide-spread meningitis; this is perfectly true, but peripheral suppuration of the brain is also very frequently found in these cases. On the other hand, a diffuse suppuration of the brain is said to be more probable when, after symptoms of general cerebral disturbances, the patient dies in a comatose condition at the end of the third or fourth week. Experience destroys these illusions, and it is quite certain that the presence of the symptoms of a localized affection affords as yet the only reliable guide to the diagnosis. These symptoms, however, will be relatively seldom present, for it is evident that only a small portion of the cortex is capable of exciting special symptoms when it is diseased. Only psychical defects of a very general nature are produced by circumscribed destructive processes in by far the greater part of the cortex. Moreover, it must be remembered that only a small number of the nerve-fibres contained in the white substance of the hemispheres are destined to conduct the sensible and motor impressions to and from the body, the office of by far the greater number being to unite different portions of the cortex. Hence, they are of great importance with regard to the rapidity and brilliancy of the psychological processes; but an isolated lesion of a system of fibres will be as little liable to cause a distinct, isolated, psychical defect, as would be a lesion of a portion of the superficies. Consequently, an acute encephalitic focus may be found, under the above-mentioned conditions, in the parietal, occipital, or temporal lobe, although no manifestations of a localized disease, no psychical symptoms—our knowledge of which, however, is still very crude—had indicated its existence. Of course, when diffuse meningitis is present, it is impossible to ascertain a more minute psychical defect.

It follows from the above that an acute encephalitis, following a non-perforating injury of the head, can run its course without our having a suspicion of its existence. A concussion of the brain, for instance, has taken place with a contusion situated in the interior of the organ (solution of continuity of the tissue with capillary apoplexies involving a variable extent of the organ), or a small splinter has been broken off from the vitreous table, and has perforated the dura and pia and buried itself in the brain (several such cases have been observed). The first manifestations are those of commotion; there are no symptoms of localized disease. The symptoms of commotion for the most part now pass away, but the cerebral symptoms do not entirely disappear; they only undergo a change. The patient is but half conscious, he is very sleepy, and is delirious now and then; it is difficult to waken him out of his peculiar condition, and he then complains of severe headache and dizziness, in consequence of which he staggers or is unable to walk; an irregular fever appears. There is no trace of the symptoms of a local disease; the pupils are equal, yet variable in size; at times they react slowly, and then there is a slight transitory dilatation of the pupil. The sleepiness and languor of the patient increases to a transitory sopor; the countenance, usually red and injected, is occasionally pale; the excited pulse sinks at times to 70 or 60. There is frequently confusion of ideas, which is followed by clearer intervals, and these again by sopor. This period, during which nothing can be diagnosed, except a marked cerebral irritation with manifold intimations of compression, which again disappear (that is, fresh interference with the circulation in the psychical organs of the brain), has a very variable duration. In some cases the symptoms are so insignificant that after a few days complete euphoria seems to be re-established, and the fears of both physician and patient are quieted. Often, indeed, the case does not come under the observation of the physician at all, for the injury appears to the patient, and perhaps really is, so inconsiderable, that he does not imagine it to be a cause of sickness ; or the sickness lasts from one to two weeks, and, on account of the changeable course and fragmentary character of the symptoms, it cannot be classed with any of the usual cerebral affections (commotio, contusio, compressio), and is then in practice generally called congestion. This designation is not incorrect, but the experienced physician will know that behind this congestion frequently enough something is concealed which, on account of its position, produces no symptoms of localized disease, but really is a focus of disease—acute encephalitis.

Suddenly, however, the above symptoms become more intense, the fever increases, but is very irregular ; the dizziness and headache become more marked, the consciousness is lost for good, and all at once the patient falls into a state of sopor, which may be accompanied by delirium, or may become at once so deep that even this obscure dream-life is extinguished. Perhaps for several preceding days vomiting has also been present. The pupils are now wide and fixed, the pulse is slow, and all or only a portion of the symptoms of compression of the brain are present. As the case progresses graver symptoms appear : rolling of the eyes, transitory divergence, sudden permanent paralysis of the abducens, of the motor oculi, or of the facialis, and in a few cases hemiparesis or hemiplegia. Convulsive symptoms are not wanting. They consist usually, not of isolated twitchings of the muscles, but of twitchings of both hands, especially of the muscles of the fingers, and analogous twitchings of the feet, or genuine clonic convulsions of the extremities. In some cases, however, a general convulsion suddenly sets in, which varies very greatly in duration and intensity in the individual cases. From these general convulsions, which are undoubtedly due to some cause that acts on the entire brain (interruption of the circulation by the pressure of the swelling focus), the partial convulsions which are confined to single groups of muscles must be accurately distinguished; they are twitchings of the muscles supplied by one facialis, which are followed by a paralysis, twitchings in one arm, one leg, or both, frequently followed by hemiplegia. These last are manifest symptoms of local disease, and it must now be clear to the blindest that the initial "congestion" was something more than a mere hyperæmia. Under these circumstances we once saw an intense chill with elevation of the temperature to 41° C. (105.8° Fahr.). The convulsions, especially the epileptiform ones, also cause an elevation of the temperature. The sopor now deepens into coma, the previously slow pulse is quickened and becomes irregular; the course of the temperature varies, but a continuous elevation until death occurs is the exception.

It is impossible to determine before the autopsy the stage in which the acute encephalitis will be found. We may find a wide-spread red softening of the cerebral substance, with the changes in the surrounding parts and the characteristic appearances in the focus itself, which have been already described; or the process may have already advanced to the formation of an acute abscess, the description of which has also been given above. When the symptoms have followed the above course, the process has started in a portion of the brain the lesion of which does not give rise to symptoms of localized disease; it has, however, gradually reached provinces of the brain the irritation and subsequent destruction of which must manifest themselves in symptoms of localized disease. We find recorded, however, a large number of cases which prove that the latter condition may be present from the very commencement.

The duration of the symptoms is very variable. Beck found an abscess of the brain already on the fifth day after an injury of the head; we found an abscess on the twelfth day. Some cases which we saw from the fifth to the eighth day were in the condition of red softening, without apparent collections of pus.

When the air obtains access to a peripheral cerebral contusion and suppuration, death occurs more rapidly, on account of the diffuse meningitis which is present as a complication in the majority of these cases.

It is very important, however, to remember that the above described encephalitic symptoms by no means always terminate fatally. We have already alluded to the possibility of recovery from contusions of slight degree. There is no doubt that a cure —that is, a demarcation of the contusion and the subsequent inflammation, and their transformation into one of the already mentioned harmless conditions—can result. This is proved by a large number of cases in which the dangerous symptoms of encephalitic inflammation that followed an injury disappeared, and the health suffered no further disturbance.

The transformation of the inflammatory focus into the so-called Durand-Fardel's cellular infiltration must be counted among the terminal stages which are, for a time at least, innocuous; also the transformation into a cyst, the metamorphosis to a callous thickening of the cerebral tissue, containing a nucleus colored by fat granules and a few hæmatoidin crystals; and, finally, a change into a yellowish-gray, atrophic, consistent residuum, which is rarely observed (Krafft-Ebing, Th. Simon). We call them innocuous, although, when the changes are extensive, a defect may exist in the psychical life of the patient, and although it is evident that special symptoms, dependent on the situation of the lesion, may also continue permanently, because they are not liable to excite subsequently any acute affection which would threaten the integrity of the brain and its functions. It must, however, be mentioned that the inflammatory process which has run its course in the brain can give rise to chronic disturbances of a diffuse character. The intimate details of this association are still entirely unknown. The chronic disturbances which are thus produced are as follows:

1. The calcification of the ganglion-cells in a portion of the cortex which is situated under the injured part of the skull (Virchow).

2. Chronic psychosis, in the form of irritable melancholia, followed by recovery (observed by ourselves).

3. A psychosis of very distinctive nature, which sets in soon after the injury, and is characterized by severe headache, dizziness, anxiety, and hallucinations. Soon afterwards, even while the above symptoms continue, the faculty of thinking is impaired, the ideas come slowly, and, finally, there is almost complete inability to think, together with intercurrent periods of excitement and constant illusions of the senses. At the same time there are changes in the pupils, which indicate great irritation of the surface of the brain. A few cases recover (one case of our own). In a number of other cases this condition of affairs continues for years, with but little variation, and at last ends in complete imbecility. These cases can be ascribed to chronic meningitis, with participation of the cortex. These lesions have been found in some autopsies (*vide* also the communications of Krafft-Ebing).

4. At a variable period after the injury (from a few weeks to a few years, although the years can only be admitted with a reservation), symptoms appear which in quality and course resemble those of dementia paralytica. The post-mortem appearances also correspond, though with many variations, with the lesions found in that disease. They never, it is true, resemble completely the pure, typical cases of dementia paralytica; but we must bear in mind the manifold diversities which even the idiopathic cases of that affection present. L. Meyer (Archiv f. Psych. und Nervenkr., S. 242) speaks of an individual who had fallen upon his occiput, and afterward suffered from a serous discharge from the ear and from deafness; the deafness was complete on the right side, and incomplete on the left; a few weeks afterward epileptiform convulsions set in, and were followed by the characteristic signs of dementia paralytica, which lasted, however, four years. The post-mortem examination showed a pachymeningitic extravasation of blood, exostotic roughness of the bone in the middle fossa of the skull on the right side, and a mottled appearance and atrophic condition of the cortex of the frontal lobes; microscopic examination revealed an extensive cell-proliferation around its vessels. Almost all authors (Schlager, Krafft-Ebing) agree that the development of the disease after an injury is very slow; our own observations

also show that the course is exceedingly protracted, and entirely different from that of the spontaneous cases.

5. After an injury to the head—this point has been emphasized especially by Krafft-Ebing—a psychical vulnerability frequently remains, which does not lead to a manifest psychosis unless some exciting cause be added. The psychical processes are, however, different from what they were before the injury. The feelings and thoughts of the patient are changed; great psychical irritability and hyperæsthesia, diminution of the power of sustained thought, severe psychical reactions after very slight irritations, change of the disposition—formerly, perhaps, distinguished for its stability and thorough balance—all place the patient on the boundary line between psychical health and disease. Such persons have no power of resistance against the causes of disease, and are often, long years after the injury, overtaken by a disease of the mind. In all these cases the prognosis is bad, but they do not by any means necessarily present the clinical history of dementia paralytica. A strong predisposition to psychical disease can consequently be created by an injury.

6. Epilepsy is a frequent consequence of the chronic changes in the skull that follow an injury. It is due sometimes to easily recognized gross changes in the skull and surface of the brain, sometimes to changes in the brain with which we are wholly unacquainted; care is necessary to avoid confounding the effects of the epileptic convulsions with their causes (atrophy of the hippocampus major, Meynert). In several epileptics that were under our observation, old depressions in the skulls were present, and in one case, on which we performed an autopsy, there was a defect in the right parietal bone; a dislocated lamella of bone a square inch in size was found under the edge of the parietal bone, with which it was united by a firm, bony mass. The dura and pia were firmly adherent to the bone and to the surface of the brain, and there was defect in both central convolutions, involving a space one and one-half inch square, in which the cortex was atrophied, so that it was only two millimetres in thickness.

7. Tumors of the brain have been known to follow injury

(Griesinger, Recklinghausen); the etiological connection is unquestionable, but its nature is unknown.

8. Diabetes. In a case that was under our observation this began immediately after a fall on the back of the head. It terminated in tuberculosis of the glands. The anatomical examination of the brain gave negative results.

These remarks are mere fragmentary notifications of conditions we are not called upon to consider in detail at this time.

For our purposes, the other already mentioned changes of the encephalitic focus, namely, the transition to yellow softening and into the capsulated abscess, are of fundamental importance. It is unfortunately at present impossible to say why, in some cases, the inflammatory focus is replaced by a spot of necrotic softening, which, as experience teaches, possesses a fatal tendency to spread at intervals, after it has remained quiescent for a considerable time; and why, in other cases, the neuroglia produces a cyst-wall which isolates the pus, and converts the acute process into a chronic abscess of the brain. We found, while investigating the anatomical lesions, that the two processes—necrosis of the tissue, and suppuration—are combined; hence we must assume, in each particular case, a predominance of the former and a cessation of the latter, or *vice versa;* this, however, still leaves the essence of the process unexplained. In both cases the processes may become quiescent for a time; the congestive fluxion, to which we refer the symptoms of cerebral irritation, and the œdematous transudations in the neighborhood of the focus, to which the varying degrees of cerebral pressure are partly attributable, cease. It will then depend on the location of the focus of disease, whether symptoms which point to a localized affection remain behind or not. The period of latency which follows the acute initial stages will consequently vary greatly in completeness.

We will return to this point when considering the symptoms of chronic abscess of the brain.

II. *Acute encephalitis, complicating affections of the petrous portion of the temporal bone* (*otorrhœic encephalitis*) *and of other bones of the skull.* It is very difficult to give an exhaustive description of the acute encephalitis which accompanies caries of the petrous bone. On the one hand, the clinical histories present very great differences in the individual cases, and on the other they are obscured by the frequent co-existence of meningitis and thrombosis of the sinuses. The complicated cases are tolerably numerous. Finally, it seems that in these cases also the encephalitis may commence without symptoms, which make their appearance only when the focus of red softening has attained a certain though necessarily still small size. Hence, it is by no means always possible to ascertain the date of the commencement of the affection. The difficulty of the diagnosis is increased by the circumstance that no bands of fibres, which are direct conductors of sensibility and motility, pass through the temporal lobe. Hence, an abscess in that lobe may attain a considerable size, and may cause general symptoms of compression before any distinct symptom of local disease arouses the suspicion of a localized affection of the brain. For this reason the acute abscesses belonging in this category, in the great majority of cases, have not been positively diagnosed.

The duration of the preceding otorrhœa is very variable; in some cases it has lasted some months, in others the otitis is congenital, and the purulent discharge from the ear was noticed immediately after birth. It occurs in individuals of all ages. A patient who came under our own observation suffered from otorrhœa for twenty-six years before it killed him.

35. A servant-girl, thirty-one years of age, was admitted April 25, 1869; of healthy family; no previous sickness; gave birth to a child a year and a quarter before, and since then has been a wet-nurse. On April 24th she complained of severe pain in the head and the left ear, noises in the ears, dizziness, thirst, chilly sensations, and sweating. A few weeks previously she had received a slight injury on the forehead, and since then had suffered from dizziness; she is said to have been hard of hearing, and to have complained of pain in the ear for several years. Purulent discharge from the left ear; total abolition of its functions; considerable swelling of the cervical glands.

From the first of May she was seriously ill; suffered from intense pain in the forehead and neck, and dizziness; from that evening complained continuously of these pains, and of pains in the left ear, and of impairment of sight. On the evening of the second day a hot mustard foot-bath was administered, which was followed by slight improvement and by a quiet night. On the third, however, the old pains reappeared, and continued until the morning of the fourth, when death took place suddenly. Motility and sensibility were undisturbed; herpes on the lips, and fetid smell from the mouth, probably due to discharge of pus through the Eustachian tube. The temperature in the axilla was, on the average, 1° R. (2¼° Fahr.) above the normal, until the morning of the 29th of April; but from that date it was normal.

Autopsy.—Dura tensely stretched and anæmic; the large veins of the pia moderately distended with blood, the small ones empty; surface of the brain dry; convolutions flattened, more on the left side than on the right, the middle lobe firmly adherent to the petrous portion of left temporal bone; on the posterior outer portion of the petrous bone the dura was firmly adherent to the pia and brain-substance over a circumscribed space, and the part of the bone corresponding to this spot was entirely softened. In the middle lobe of the brain, an inch and a half behind the anterior border of the lobe, was a focus one and a half inches long, that reached to the surface, and was filled with a stinking, greenish mass. The cerebral tissue surrounding it was softened, grayish-black in color, œdematous, bluish, and of gelatinous consistency (bläulich-sulzig), and in several places was riddled with hemorrhages. The rest of the brain was very pale and œdematous (Wendt).

36. A man, forty-three years of age. On January 20, 1855, he experienced a strange feeling of lightness in his head, as if he were lifted from the ground; could not understand those standing around, and could not speak to them. At home shortly afterwards he had a convulsive attack, in which he lost consciousness, but recovered after a few minutes. In the afternoon there was a repetition of the attack (sensation of being suspended in the air, with subsequent loss of consciousness and convulsions); recovery. On the 23d and 24th there was restlessness and sleeplessness at night. On the 25th at night, severe headache in the temporal region; restlessness. On the 26th, extension of the headache to the upper part of the neck; irregular pulse, often only 50; unsettled state; coma; death during the morning.

Autopsy.—In the posterior part of the middle lobe on the right side a recent abscess, almost as large as an orange, which reached to the convolutions covering it (Gull).

37. A man, twenty-nine years old; otorrhœa on the left side since he was three years of age (some acute disease at that time, whose nature cannot be ascertained). Five years ago had typhoid fever. For the last three years has had chronic cough, with emaciation and loss of strength; had hæmoptysis once two years ago; at present has constant cough, purulent expectoration, high fever, and night-sweats.

September 28th.—Otorrhœa on the left side; membrana tympani almost entirely wanting; granulations in the tympanic cavity; the tuba closed; hearing entirely lost. Infiltration of both apices, especially of the left; amphoric and bronchial respiration; ringing rhonchi. Since the evening of the 24th has had great dizziness and a slight headache, for which he came under treatment. Temperature 38.2° to 38.8° C. (100.76° to 101.84° F.). Pulse 100 to 106.

September 26th.—Sleepless night; restless; more headache; buzzing in the head; sleepy appearance; movements slow and heavy; increase of headache; pupils narrow, reacting badly; innervation everywhere perfectly normal; no disturbance of sensibility; gait somewhat staggering, but there is no paresis.

In the evening all the appearances were the same, but the headache was so great that bloodletting was added to the derivatives and the ice-bladder. At nine o'clock in the evening a sudden and short epileptic attack; the consciousness was lost for a quarter of an hour, and then the complaints of general headache began again. Temperature 38.4° to 38.8° C. (101.12° to 101.84° F.). Pulse 100 to 98.

September 27th.—More torpid condition of mind, together with complaints of intense headache, ringing in the head, and dizziness. Gait tottering; when he walks he lets the head droop forward; every contraction of a muscle in the upper part of the trunk and the neck increases the pain in the head. Pupils narrow, react badly; muscles of the eye, face, tongue, arms, and legs normal, but all the movements are weak and slow.

Vomiting; temperature 38.6° C. (101.48° Fahr.). Pulse 82, full.

During the day he had three epileptic attacks, each of about five minutes' duration; the coma lasted longer after the second than after the first attack, and after the third it continued until death. Temperature 38.8° C. (101.84° Fahr.). Pulse full and irregular; no more vomiting; pupils medium size, do not react; no paralysis can be discovered. The diagnosis then was meningitis purulenta.

September 28th.—Coma continued during the entire night; an examination of the retina made in the morning revealed congestion of the retina, such as occurs in meningitis. Snoring, slow respiration; pupils medium, but do not react; no spontaneous movements are made, all the movements being reflex. Temperature 39° C. (102.2° Fahr.). Pulse rose again to 92, small.

In the evening death during coma. Temperature 39.2° C. (102.54° Fahr.). Pulse 112, irregular.

Autopsy.—Abscess in the left temporal lobe the size of a pigeon's egg; dura and pia united to the surface of the brain over an area of a square centimetre, and infiltrated with pus; around this old adhesion commencing purulent meningitis. The abscess had no capsule, the tissue surrounding it was softened, and presented a red border a line in breadth, with a few small apoplexies. General anæmia of the brain and great general œdema. (Case observed by ourselves.)

In other cases of equally rapid course, some symptoms of a localized disease are present, *e.g.*, weakness of the left side (Gull, Ollivier).

In these cases the acute abscess of the brain runs a very rapid course. It is difficult to draw with accuracy the boundary line between acute and chronic abscesses, for in many cases no accurate description of the abscess is given, and it is very doubtful whether the commencement of the symptoms was simultaneous with the beginning of the change in the brain. Cerebral abscesses have been found especially in the temporal lobes, the existence of which had not been betrayed during life by a single symptom; we have ourselves observed one such case where we had no suspicion of a cerebral affection (see also Heusinger, Virchow's Archiv, XI.). The duration of the cases which we include in this category with the above reservation, varies from four to about twenty-four days. The earliest changes in the brain which lead to acute abscess have as yet very rarely indeed come under observation, and are as good as unknown; we have already stated that we believe ourselves justified in the opinion that some of these abscesses never pass through a distinct stage of red softening, but are collections of pus from the very commencement.

The majority of cases present the symptoms of a rapidly initiated and increasing pressure on the brain, interrupted by convulsive attacks of a general nature, which usually hasten the appearance of coma. The course of the fever is not constant; the pulse conforms to the course of the cerebral pressure.

Few cases present symptoms of localized disease during their course. The abscess is usually located in the temporal lobe, and seldom reaches the important centres lying above and toward the median line.

The cause of death, except in a few cases of perforation and complication with meningitis and thrombosis of the sinus, is to be found in the cerebral compression exerted by the growing abscess and by the great œdema of the brain.

The individual symptoms may now be briefly described:

In the great majority of cases the first symptom is the intense headache, which increases from hour to hour, and is interrupted only by short intervals. Accompanying it we find recorded in the different cases: dizziness; ringing in the ears; disturbance of the power of thought and of memory, with mild delirium, even in the earliest stages (when complicated with meningitis); and in one case, already cited, "a strange feeling of lightness in the head." Vomiting is frequently one of the earliest symptoms; no symptoms of localized disease, even in the pupils; nothing but contraction and a slow reaction is discoverable. In acute abscess the condition rapidly becomes threatening; the headache becomes more and more intense; at times the patient is unconscious or delirious, and a more or less intense cerebral compression is quickly produced. At this time, or even earlier, an epileptiform convulsion occurs, which is followed by sopor. This, however, does not occur in all cases, by any means, but is observed in somewhat more than half the cases, if we may be allowed to deduce a statistical estimate from the small amount of material at hand. Convulsions, confined to one side of the body, or to the parts supplied by the facial nerve, are rare; such convulsions are, of course, to be classed among the symptoms of localized disease. The convulsive attack may be single, or may be repeated several times. The observer is surprised in most cases by the rapidity with which the fatal termination sets in. The pressure on the brain increases rapidly until deep coma is produced, during which death takes place. In a few cases the death was preceded by basilar symptoms, which were due to purulent meningitis of the base. This latter, as well as thrombosis of a sinus, is especially liable to influence the course of the affection in such a way that it is impossible to accurately recognize and differentiate the different conditions.

In this series of cases, therefore, the abscess can only be recognized from its characteristic course and its combination with general convulsions.

The other series of cases begins in a similar manner; for some reason, as yet unexplained, it is free from convulsions, but is attended by symptoms of localized disease. The latter are due to the fact that the abscess, in the course of its rapid enlargement, grows upward and involves the base of the nucleus lentiformis as well as the outer surface of the adjoining peduncle. The result of this is a hemiplegic disturbance, which, however, did not terminate in complete hemiplegia in any of the cases that are known to us. Disturbances of sensation have also resulted from it (Ollivier), as well as paresis of the oculo-motorius and facialis. These cases, also, are characterized by an exceedingly rapid course.

A third series of cases finally presents a combination of local paralyses with epileptiform convulsions; these cases, unfortunately, are not all pure, but are frequently complicated with meningitis or thrombosis of a sinus—a condition which, under all circumstances, renders the diagnosis doubtful. The physician would certainly not diagnosticate thrombosis of a sinus unless he has a sure basis for his diagnosis (local congestions). The often realized impossibility of a certain differentiation of meningitis from an abscess of the brain, which is attended neither by local symptoms nor by convulsions (such cases have been seen), renders further consideration of the subject useless.

III. *Acute encephalitis of a purulent character in connection with affections of the lungs.*—The nature of the pulmonary affections has already been described; bronchiectasis, with putrid secretion, is the most important. The duration of these not very rare affections is rather variable; the boundary line between acute and chronic abscess is sometimes passed, so that in the more chronic cases abscesses may be found which possess pretty distinct capsules. In the great majority of the cases, however, they have not gone through the characteristic course of chronic abscess of the brain; the existence of a period of latency cannot be ascertained, the course of the affection being, for the most part, progressive from the beginning to the end.

Here, again, the characteristics of some of the post-mortem appearances are such that we are compelled to believe that some of these abscesses are not preceded by a stage of red encephalitis, but constitute from the very commencement small collections of pus.

The following case, observed by Biermer and published by R. Meyer, is a good example of the affection:

38. A man thirty-six years old; entrance to hospital July 4, 1864; death July 8th. Three years previously he had pneumonia, and since then had cough, with profuse, stinking, purulent expectoration; fetor ex ore. For some time had suffered from exhaustion and languor, without much emaciation. On the 26th of June he was seized with a troublesome twitching and formication in the right hand, which became stiffer and less movable. In the course of the day both symptoms became more marked, and extended also to the right foot; there was no real paralysis, but some limping. These symptoms continued until July 1st. During the night of July 1st he had a chill, accompanied by confusion of ideas, cloudiness of the sensorium, partial paralysis of speech (aphasia?), and motor paralysis of the right side on awaking. Since that time he has been slightly delirious; passed his

urine involuntarily, bowels constipated, no vomiting, but had intense frontal headache. Never met with any injury.

July 5th.—Left pupil somewhat dilated; both bulbs retain normal power of motion; paresis of the right facialis; tongue straight. Fetor oris increased by coughing; stinking, greenish, confluent sputa. Hemiparesis of the right side. If the arm and leg be touched while his eyes are closed, he constantly confounds the right with the left side; but when his eyes are open, he recognizes his extremities perfectly well. Night quiet; no delirium; urine passed in bed; psychical processes slow, but there is no real loss of consciousness. Urine normal. Constipation. Dullness and bronchial respiration under the right clavicles. Temperature 37° C. (98.6° Fahr.). Pulse 52, pulsus cephalicus. Liver normal; spleen somewhat enlarged.

6th.—Pulse 52; evening 56. Sensorium inactive, otherwise no change.

7th.—Evening, at 7 o'clock, convulsive attack; patient bends the head backward; breathes deeply; contracts and extends the left arm convulsively; right side motionless; reflex excitability increased on the left side, so that the entire side jerks convulsively when slightly touched. Duration five minutes. Speech afterward to a great extent lost.

At 8 o'clock a second attack: tetanic convulsions of both lower extremities, of the upper extremities only the left taking part in the convulsions by irregular movements. Complete loss of consciousness; both pupils greatly dilated, the left more so than the right; duration three-quarters of an hour, followed by sopor, snoring respiration, and loss of consciousness until 10 o'clock.

Then the pupils contracted again; the patient swallowed, and this act was followed by still another attack of convulsions involving the left side of the face and the left arm. Ten minutes later there was a similar, but less violent attack. This was followed by sopor, with dilated pupils, the left being the larger. Slow pulse; temperature not elevated; no sweating; continuous slight clonic convulsions in the left arm.

About half-past twelve o'clock, repetition of the clonic convulsions, which subsequently recurred—at first, every quarter of an hour; later, every half-hour—with increasing severity. They involved especially the left side, the right half of the body taking part only in a small degree. Between these attacks, deep lethargy, with snoring respiration.

At two o'clock, collapse; tracheal râles. At four o'clock, still another tetanic attack; respiration deep and slow; pulse very quick, and soon became uncountable. From four o'clock, cyanosis; increasing collapse; and, at six o'clock, death.

Autopsy.—Veins of the pia tensely distended; the membrane, at several points of the left convexity, adherent to the brain. Dura and sinuses normal. Brain soft; in the left frontal lobe distinct feeling of fluctuation; similar, but less distinct feeling in the right posterior lobe. At the apex of the frontal lobe, on the left side, the surface had a grayish-yellow color. At the posterior end of the right hemisphere, a little pus welled out from an accidental fissure. The medullary substance of the left hemisphere, in front, was in a condition of marked white softening. The posterior cornua of the ventricles were greatly dilated; the fornix was softened; and in the left frontal lobe there was an extensive sinuous abscess, filled with greenish, slimy pus. This abscess extended downward as far as the under periphery of the cerebrum, and backward toward the lateral ventricle, almost to the corpus striatum. Wall smooth; the tissue in the immediate neighborhood indurated, cutting like cheese. A second abscess, the size of a hazel-nut, was found in the middle portion of the left hemisphere. In the right hemisphere there were three smaller abscesses: one, as large as a plum, in the apex of the posterior lobe; another, as large as a pea, situated far back in the middle lobe; and the third, as large as a cherry-stone, in the apex of the frontal lobe. No injury to the skull.

Left lung presented solidification in the lower portion of the upper lobe; pleura thickened; parenchyma retracted. On section, a cavity was found in the neighborhood of the root of the lung, the walls of which were studded with filamentous, ragged, steel-gray remnants of old connective tissue; numerous bronchial tubes open into it. At these spots the wall was rendered smooth by newly formed connective tissue, and the bronchial walls were directly continuous with it.

The patients who die of this form of abscess of the brain have almost always previously suffered from some disturbance of nutrition, to which must be ascribed, not the cerebral affection, but the primary pulmonary affection. This primary pulmonary affection is often a bronchiectasis of from ten to fifteen years' duration, attended by frequent exacerbations of the bronchitis, in which the putrescence of the secretion is at times more, at other times less marked.

The initial symptoms of the brain affection are different. They are not necessarily connected with a particularly marked fetor of the sputum. In one case seen by us, but unfortunately not completely observed, the patient was relatively free from pulmonary symptoms when the brain symptoms began. The first signs are different in different cases. In some cases they are those of a developing local affection, with great arterial congestion of the brain, viz., severe headache, usually confined to the forehead; dizziness, alteration of the psychical processes, slight somnolence, occasionally slight delirium, and, in addition, some symptom which indicates an active irritation of some of the motor fibres of the corona radiata and their terminations in the cortex; unilateral convulsions in an arm or leg, or both; dysæsthesia (formication and the like), or a slight diminution of the acuteness of sensation. Such symptoms, occurring in a patient who is suffering from a chronic pulmonary affection (phthisis, too), must arouse suspicion of an encephalitis. In other cases the focus is not situated in the place mentioned, but in some part which, when diseased, does not produce special symptoms. The initial symptoms of irritation are then in no way characteristic, and are just as liable to awake the suspicion of a diffuse superficial affection. A third series of cases, finally, presents at the commencement no general symptoms, but only the symptoms of a localized disease (the above case). Here we may choose between two opinions: either the local affection begins, not as red softening—which under all circumstances causes violent disturbances of the cerebral circulation—but as a small collection of pus, surrounded by a very delicate inflammatory zone, which rapidly enlarges, and involves neighboring systems of fibres; or the focus has been developed very slowly and imperceptibly (hardly altogether without symptoms, but the symptoms were underestimated), and suddenly, under some unknown conditions, takes on a more rapid growth. This is apparently confirmed by the fact that one set of cases does not possess a capsule, while in another set a capsule of considerable thickness is found. The initial special symptoms are convulsions, confined to special sets of muscles, or a slight hemiparesis, which is not preceded by convulsive movements, although in the majority of cases it is ushered in by a convulsion.

The time which now elapses before the appearance of severe symptoms is always very short; if any one prefer in the above mentioned and similar cases to designate this relatively free interval a stage of latency, he is at liberty to do so.

As the affection progresses, indications of a severe disease of the brain are rapidly produced. In one class of cases the combination of general and local symptoms continues:

The confused state of the intellect and delirium become more intense; the former predominates in the majority of cases, and leads to conditions of intense sopor, the patients lose consciousness of place and time, and become uncleanly.

The headache and dizziness increase rapidly; the former attains an unendurable degree, and the latter interferes with the movements, especially of standing and walking.

The local symptoms are usually continued as symptoms of active irritation ; the previous localized convulsions are repeated, and lead to paralysis ; convulsive movements of the eyes often occur, and twitchings of the muscles supplied by the facialis, on the side corresponding to the affected half of the body. In some cases of abscess of the brain situated on the left side, disturbances of the speech occurred, which, according to the descriptions, were undoubtedly aphasic.

The type of the convulsions may now suddenly change ; epileptiform attacks set in, which vary in duration and intensity, and always cause a considerable aggravation of the otherwise fluctuating course.

The increase of the intracranial pressure is revealed by the characteristic symptoms, which are sometimes, however, only partially developed ; vomiting is frequently absent ; the retardation of the pulse is also often wanting, or is present only at intervals, while in other cases an evident pulsus cephalicus is observed ; toward the end of life the pulse always becomes small and frequent. Constipation is common. The retina presents the signs of venous congestion, which are more marked on the side of the abscess ; this corresponds with previously described opinions about its origin.

The temperature is very inconstant. In one of our own cases the commencement of the affection was attended by a chill with considerable elevation of the temperature, but the subsequent course of the affection was marked by a low temperature. In other cases, febrile conditions of different, often very considerable intensity, exist, but in course and degree they are entirely inconstant. In some cases (Meyer) true chills have been observed during the later period of the affection.

Now and then (see above case) the variations in the local symptoms enable us to diagnose multiple abscesses of the brain, or at least an affection involving both sides of the organ. If hemiplegia of one side be produced by an acutely progressive cerebral affection, and later on evident, localized convulsions of the other side occur, the conclusion that a similar process has begun in the other hemisphere is justifiable. This point has been already turned to account in the diagnosis of a superficially situated cysticercus.

The general epileptiform convulsions, during which the pupils are usually dilated and fixed, must not be ascribed to local irritations; their genesis is to be found in disturbances of the circulation in the medulla oblongata, which are undoubtedly dependent on the local affection, but which at present cannot be accurately defined. They might very conveniently be ascribed to compression of the medulla (pressure of the focus plus œdema of the brain), were it not that they often appear at a time when the psychical functions are not yet very much altered; consequently, before the pressure has reached such a grade as to cause a total compression of the vessels of the cortex.

In a second series of cases no symptoms of localized disease are observed during the entire course of the affection; the symptoms are those of a disease of the brain, characterized by gradually increasing intracranial pressure—a disease undoubtedly of inflammatory nature—a disease which, finally, entirely prevents the access of blood to the cortex and the ganglia. Indications of epileptiform convulsions appear, but these fragments (trembling of the muscles of the fingers, and the like) give no conclusions.

If the nature of the lung affection is not positively recognized, the cerebral affection can easily be mistaken for meningitis tuberculosa; several such mistakes will be found recorded in the literature of the subject.

Finally, in the third series of cases, only the symptoms of localized disease are present at the outbreak of the disease; but general symptoms subsequently set in, and the course of the affection is then similar to that described above.

The duration of the process varies between five and twenty-eight days, or we may say between one and four weeks. There are, however, transitions to the picture of the chronic abscess of the brain, which will be briefly mentioned later on. This variety of encephalitis also can, in very rare cases, become chronic; that is, after the appearance of the first symptom, the affection does not necessarily progress uninterruptedly to a fatal termination, but a free interval (period of latency) may exist, which is followed by an acute terminal stage. The abscesses, then, evidently belong to the chronic variety.

Our anatomical knowledge of the genesis of abscess of the brain under these circumstances is very meagre; the views of Boettcher have been already detailed.

IV. *Acute encephalitis in pyæmia.*—It corresponds in many respects with the traumatic variety, and its erection into a special variety is only justifiable because it occasionally comes under observation as a lesion of non-surgical pyæmia (puerperal pyæmia, endocarditis ulcerosa). This variety of acute abscess of the brain (it is usually multiple) is very rarely seen in its first stages. The abscesses at first consist of scattered, often very small, but numerous foci of red softening, which are located by preference near the periphery of the brain; when they are quite recent, they present the appearances of hemorrhagic infarcts, which, however, are undoubtedly due to specific emboli. When the cases are a few days older, small abscesses are found, which do not possess investing membranes, but are sharply circumscribed and surrounded by an exceedingly small zone of true inflammation. If only one abscess exists—a very rare occurrence under these circumstances—and the primary disease be curable, it may be transformed into a chronic abscess. The affection, however, in most cases runs a very rapid course, which is attributable partly to the nature of the primary disease. Since the latter is itself capable of producing a severe affection of the sensorium, the abscess may be entirely overlooked in cases where it does not involve any important motor or sensory provinces of the brain, and does not attain any great size; the commencement at least of the disease of the brain is generally entirely masked. The symptoms are very confused, because the foci are usually multiple, and are located in very different spots, though, in general, close to the periphery; hence, the clinical picture is necessarily somewhat confused. The most prominent symptoms are those of a constantly increasing swelling of the contents of the skull (cerebral compression), and an alternation of sopor and delirium. Usually there are several chills, which, however, seem rather to depend on the primary affection. Convulsions occur in this form, but they are by no means the rule; in the few cases observed by ourselves, they did not occur. Here, too, the signs of congestion of the retina are in the beginning more marked on the side corresponding to the abscess of the brain.

V. *Encephalitis around pre-existing lesions in the brain.*—We have to consider here tumors, necrotic softenings, and extravasations of blood.

a. Necrotic softening from thrombosis of an artery and embolism. This is not the place to discuss the symptoms of these processes in the vessels. We shall merely mention that the consequent hemorrhagic infarct is a localized lesion which, when located in the proper place, may cause manifold special symptoms, especially unilateral disturbances. In the critical investigation of the secondary inflammatory processes around hemorrhagic infarcts, Hasse has surpassed all other authors, and has cleared up to a great extent the existing views upon it. He states that a peripheral inflammation takes place around the primary focus, which by degrees succeeds in giving an entirely different appearance to the focus produced by the hemorrhagic infarct. Very recently we have had an opportunity for the first time to make investigations which enable us to form a judgment ourselves upon this subject. We certainly do not find the encephalitic process around every hemorrhagic infarct; evidently it depends upon certain hitherto unknown qualities of the latter whether a distinctly recognizable inflammatory reaction takes place in the circumference or not. When it occurs, a small red areola, studded with small capillary extravasations, is formed around the hemorrhagic infarct; this is surrounded in turn by a yellowish zone, in which the elementary degenerative changes described by Meynert can be seen; and this, finally, by a more or less extensive zone of œdema of the brain. In the red zone there is migration of cells. It is now clear that, at this period also, the possibility of an aggravation of the entire process terminating in suppuration cannot be denied (Hasse). Although we have observed a large number of cases, we have, however, never been able to discover anything like that in connection with non-infectious emboli, and much less in connection with simple thrombosis. The case is entirely different when we have to deal with infectious emboli; these cases, however, belong under the categories already treated of. With regard to the retrogressive changes, we have in two cases seen sclerosis of the inflamed zone; a few remarks concerning them have already been made under the head of the pathological anatomy. In both cases the capsule was entirely closed; around it was a very delicate macroscopically inperceptible zone of yellow softening, like that which accompanies abscess of the brain (Rindfleisch). There was no trace of pus, but the usual contents of old necrotic foci. A more frequent termination, however, is the following: the inflammatory process around the focus gradually ceases, and is replaced by reparative processes, which transform the affected spot of the brain in time into a variously shaped cyst or a cavity with connective-tissue septa, or, finally, into multiple small lacunæ with usually serous, slightly cloudy contents. We agree most decidedly with the opinion of Bamberger, that in a focus due to embolism or thrombosis, these secondary changes, which, in the majority of the cases, cause an increase in the size of the original focus, may cause complete destruction of the affected vessel. The nature of the process can then only be determined from the other lesions in the body. Finally, an apparently quiescent focus, which is undergoing the reparative changes, may suddenly begin again to increase peripherally. In such cases we are usually disposed to assume a fresh attack of inflammation in the surrounding tissues. The actual existence of such an inflammation we have hitherto been unable to demonstrate with certainty; in favor of it, however, speaks the fact that in the surrounding tissue an œdema, very similar to the inflammatory œdema, is found, which, moreover, in the majority of cases, must be considered the chief cause of death.

b. Apoplexy.—The inflammatory processes of the brain-substance around apoplectic foci are of great importance. Here again the intensity of the process is susceptible of very great differences. We are not able to determine positively whether, in certain cases, for some unknown reasons, the consecutive irritation of the surrounding brain-substance may be entirely wanting; it almost appears as if this may be the case. In a very great majority of the cases, however, we find, within a very variable period after the apoplectic attack, a zone of red softening around the primary focus, the elementary processes of which entirely correspond with those of true inflammation. It is not difficult to demonstrate the migration of cells in it. Here, too, the terminations are entirely similar to those already mentioned. Some authorities state that they have observed suppuration. Hasse says that he has never seen it, and we have ourselves never come across it any more than in the simple infarcts. On the other hand, in some cases which were undoubtedly apoplexies, we have distinctly proved the formation of a secondary sclerotic capsule from closely packed and excessively numerous connective-tissue corpuscles. The further transformations are those already briefly mentioned. It is especially important to know that the secondary encephalitis may attain such dimensions that it can alone put an end to life. We often find an extensive œdematous infiltration of the surrounding tissue, in which but scanty evidences of encephalitis can be found, while the necrotic processes are unusually prominent. Hence, we have here also, in consequence of the very peculiar characteristics of the nervous and the glia tissue, a concurrence of the two previously described changes, and it depends upon unknown circumstances which of the two assumes the upper hand. According to Hasse, secondary hemorrhages occur in the circumference of the primary apoplectic focus.

c. *Tumor.*—The secondary softening around a tumor is of the very greatest importance; for it not only gives rise to a whole series of symptoms which begin suddenly, and which, when the tumor is of slow growth, can scarcely be attributed to its direct action, but it also often enough becomes a direct cause of death, in consequence of the acute character it assumes under conditions not yet accurately ascertained. In general we may say that the secondary changes are most marked in connection with the tumors which grow the quickest—the carcinomata. These can easily be separated into several subdivisions, in accordance with their gross anatomical appearances. A more accurate examination, however, shows that the processes cannot be sharply differentiated from one another, but are blended together in manifold combinations.

a. Simple softening and œdema of the circumference. Here the histological processes of necrosis can be demonstrated over a wide space ; the œdema can be ascribed to a simple transudation from the vessels, consequent upon a marked interference with the circulation that is caused by the pressure of the growing tumor (retardation, or even thrombosis). When the pressure is so great that the circulation in certain parts of the circumference is completely stopped by the compression of the capillaries, yellow necrosis is the immediate result. In these cases, however, according to our own experience, genuine inflammatory disturbances are also often present. The little extravasations of blood that accompany the encephalitis are here a very unreliable diagnostic point, for they may be due to numerous other causes (obstruction to the venous current, degeneration of the vessels), and do not by any means always indicate active congestion. The only absolutely certain proof is the demonstration of a more or less extensive migration of cells, and this can in reality be furnished. Therefore we believe the process that is observed in the circumference of the tumor to be an encephalitic one.

b. Predominance of capillary and larger extravasations. We have just given our opinion regarding these ; we hold that the fatty degeneration of the vessels, which is so frequently observed, is the chief cause of them.

c. Genuine, wide-spread, encephalitic, red softening. It presents all the characteristics of the process : the numerous ruptures of the vessels, the migration of the cells, the necroses in the inflamed tissue, the accompanying wide-spread œdema of the brain.

d. Finally, suppuration around tumors is rather rarely met with (carcinoma, Hasse ; cholesteatoma, Virchow ; glioma, Webber ; tumor on the convexity of the left hemisphere, Lutz ; sarcoma in the interior of the hemisphere, observed by ourselves, etc.). It is then usually surrounded by a border of red softening, of variable width, and the secondary changes in the neighborhood are the same as those which have been already repeatedly mentioned.

With regard to the symptoms of this form of encephalitis, the following may be stated :

a. Apoplexy.—Consecutive disturbances are most frequently observed after a genuine apoplectic attack, for the disturbance is then more intense than after a hemorrhagic infarct from plugging of a vessel. First of all, we must not confound the aggravations of the condition and the deaths which occur during the first hours (from twenty-four to thirty-six) after an apoplectic attack, with the encephalitic disturbances. For instance, a few hours after awaking, the intellect becomes clouded again, followed by more vomiting and coma, which can be modified in various ways by convulsions, a clinical picture which resembles very closely that of encephalitis. Or the convulsions may be absent, and, after consciousness has been partially regained, the coma is simply re-established. In such cases we must not infer encephalitis, because a rational basis for its diagnosis is indispensable, and this is not given in the above situations. Under these circumstances we usually find perforation of the apoplectic focus on the surface, or into the ventricle, or a fresh hemorrhage, which followed close upon the original one.

Other symptoms which follow the hemiplegia must, however, be ascribed to the consecutive inflammatory processes. A patient has made a good recovery from an apoplectic attack, but several days afterwards begins to have fever; pains in the head appear, or a dull existing headache becomes aggravated; slight wandering of the mind and confusion of ideas set in, soon followed by transitory and not very deep sopor; slight delirium; the pulse rises, and for a time corresponds with the variations of the temperature. These general symptoms are very soon followed by local symptoms, the well-known secondary contractures. These signs of irritation in the injured portion of brain do not appear in any stated order. One patient has slight tremor in the paralyzed arm and leg; another has only a contracture of the flexors; while in a third these conditions alternate. Cases which are apparently identical in the severity of symptoms and in the anatomical basis are distinguished by the different degree and duration of these subsequent conditions. Not unfrequently the temperature of the paralyzed side is considerably elevated, and anomalies of the sweat secretion are observed. The condition may become threatening, the somnolence increase to a sopor lasting several days, and the pulse become an evident pulsus cephalicus. Death may occur with the symptoms of increasing pressure on the brain, a gradual increase in the pulse-rate, and a variable temperature. Encephalitis surrounding the focus, with wide-spread œdema of the brain, figures as the cause of death.

These symptoms in the individual cases present the most manifold variations. Oftentimes they are merely foreshadowed; often recovery sets in after three or four days of the secondary sickness; we have seen a few patients recover after from eight to ten days of intense sickness, so that the hemiplegia was the only remaining symptom of local disease. Unfortunately, however, these relative recoveries are frequently incomplete, some symptoms remaining which prove that the encephalitic process has not entirely ceased, but continues, in a more or less intense degree, for some time. A chronic headache remains, and there are frequent intense attacks of dizziness; fever is certainly absent, but a series of congestive attacks, which occur from time to time and are always dangerous, are easily recognized. Each attack can cause new convulsions in the paralyzed limbs, while a permanent contracture of these limbs is left behind; generally also there is a very troublesome painfulness of the paralyzed limb; the pains vary in character, and are situated either in the joints, or in the bones, or in the skin and muscles. They are only partially dependent directly on the encephalitis; their more detailed description, as well as the consideration of the various trophic changes, does not belong here. The secondary encephalitis is also the chief cause of the atrophy of the brain observed in many of these patients, which unavoidably leads to psychical destruction, to dementia. The nature of this general disturbance of the nutrition of the brain is yet very obscure. Relying on a series of anatomical investigations, we do not hesitate to assert that the disturbance of nutrition is much more extensive than one is disposed to assume from the usual post-mortem appearances. Throughout extensive portions of the cortex, over an apoplectic focus, which is surrounded by an encephalitic and an œdematous zone, the elementary structures have undergone disturbances of nutrition, which can only be ascribed to the long-continued compression and a marked interference with the circulation; even the opposite hemisphere presents in the cortex and neighborhood similar though less intense changes, a fact which is in accord with the present views on the diffusion of cerebral pressure. These changes are not inflammatory, but are, to make use of an expression already often employed, of necrotic nature. A great number of the cellular elements of the tissue, ganglion-cells, neuroglia nuclei, endothelial cells from the lymph-vessels, connective-tissue corpuscles from the walls of the vessels, are undergoing transition into granule-cells, or are already wholly transformed into such. This shows itself in the ganglion-cells as a change in the protoplasm, which is observed in connection with a number of degenerative processes of the brain of chronic or acute nature, and which has been wrongly held to be invariably characteristic of dementia paralytica.[1] The protoplasm becomes coarsely granular, the granules collect together in masses, between which a more fluid substance appears to exist; the nucleus obstinately resists the change. Gradually the entire contents of the cavity in the tissue, which has now replaced the ganglion-cell, can be reabsorbed, and the cavity contracts around the nucleus. What finally becomes of it we are unable to determine with certainty. We must, however, put it down as certain that very considerable portions of the brain may in this way be entirely deprived of their ganglion-cells. The production of the granule-cells is most active in the white substance in the œdematous zone. We repeat again, that every long-continued compression of the brain may cause these disturbances of nutrition; if it be intense and wide-spread, which will depend upon a number of accessory causes (age of the individual, power of the heart, quantity and quality of the blood, condition of the walls of the vessels), a *restitutio ad integrum* is no longer to be thought of. The duration of the cerebral compression will of course exert an essential influence on the prognosis, and perhaps still more important in this respect is the presence or absence of the already mentioned auxiliary causes. When the latter are present to a marked extent, it is permissible to ascribe the changes to a number of simple, congestive, long-continued hyperæmias of the brain (a view which we personally long opposed), particularly because the general increase of the intra-cerebral pressure during congestion would seem to necessitate a retardation of the capillary blood-current. Cramer's observations (Dorpat. Diss., 1873) show that this is at least highly probable. The influence of inelastic vessels has been mentioned before.

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[1] We formerly entertained this opinion ourselves, but have learned to regard this change in the elements of the cortex as a disturbance of nutrition of much more general nature, for we have observed it in a number of degenerative processes of the cortex, which had positively nothing to do with the characteristic processes of dementia paralytica. Hence, when it accompanies the latter disease, it must be regarded as a mere secondary change, as the expression of arrested nutrition.

The encephalitis around large apoplectic foci leads in a few rare cases to the development of a sclerotic connective-tissue capsule, which, however, bears but a very slight resemblance to the capsule of an abscess. It seems to cause almost constant irritation in the spot where it is located, for in several cases of this kind we observed frequent contractions (tremors) in the half paralyzed arm and leg for several weeks, the sensorium being relatively free, until finally a further aggravation of the acute encephalitis, with œdema of the brain, put an end to life.

Frequently enough, in old and debilitated individuals with diminished heart-power, an apoplexy is followed not by an encephalitis, but by a wide-spread necrosis of the surrounding brain-tissue (maceration)—a yellowish-white softening. We are not in a position to decide whether in such cases there is a concurrent encephalitis. The recovery from an apoplectic lesion is only temporary, or is not complete; the psychical powers are prostrated; the limbs that are not paralyzed become weak; the paralyzed limbs become cold and œdematous; the pulse becomes small; the temperature is below the normal, and death occurs in consequence of increasing weakness of the heart.

b. Thrombosis and embolism.—It is well known that the differential diagnosis of hemorrhagic infarct from apoplexy is surrounded with manifold difficulties; a number of diagnostic points that were thought to be valuable, had to be discarded when the statistics became sufficiently numerous. Nevertheless, there are many pure cases of senile encephalomalacia and of embolism in young persons, which can be recognized with absolute certainty.

In such cases also it is not difficult to understand the encephalitic symptoms that appear after the lesion ; it must be remembered, however, that they seldom attain such intensity and extent as in the analogous process in apoplexy.

In senile encephalomalacia the secondary encephalitic symptoms of irritation are in general those already mentioned ; they consist of the signs of an increased intra-cephalic pressure on the one side, and of those of an irritation in the parts surrounding the focus on the other. The explanation does not need to be repeated here. The clinical picture is usually modified by the fact that the affection generally attacks old people with rigid cerebral vessels, on the one hand, and weakened heart-power (fatty heart) and manifold senile changes of other organs, on the other. The inflammatory reaction is slight, and in not a few of the cases that came under our own observation it was almost entirely imperceptible ; the tendency is to a rapid psychical destruction without active symptoms of irritation, especially when a certain degree of senile atrophy of the brain had pre-existed. Under such circumstances the results of the congestive compression of the brain are less apparent. It frequently happens that, after the formation of a focus in the brain, a febrile condition of drowsy delirium with tendency to somnolence continues for a time, and either finally develops into complete permanent imbecility, or gives place to a condition of relative integrity. A restitution of the mental faculties ad integrum is a rare occurrence ; frequently a condition characterized by weakness of the memory, irregular and groundless changes of temper, tendency to angry outbreaks, or to tearful and sentimental emotions, is permanently left behind, which after a shorter or longer duration ends with the development of a new focus of softening in the brain. These general symptoms of a disturbed circulation in the cortex are frequently, however, not the only symptoms. Quite often, under the influence of the inflammatory disturbances in the neighborhood of the primary focus, fainting fits of a more or less severe nature occur ; every temporary congestion can, under the circumstances mentioned, lead to a transitory loss of consciousness. Partial convulsions of the half-paralyzed limbs also occur, and they tend to make the diagnosis difficult, especially when accompanied by somewhat severe headache. We observed this especially in two cases in which the encephalitis had led to the formation of a sclerotic zone, like that which is observed in apoplexy. Finally, general convulsions occur in some rare cases, and probably depend on marked disturbances of the circulation in the medulla oblongata.

The encephalitis in the parts surrounding the primary focus has a great tendency to cause an enlargement of it. This is the reason why we often see partial convulsions of the paralyzed side followed by complete paralysis, or paralysis of only one extremity subsequently followed by paralyses of the others. At last a more wide-spread inflammatory affection frequently leads to a rapid death, through a wide-spread, intense œdema of the brain.

The symptoms of inflammation in the neighborhood of embolic infarcts of young individuals may be entirely analogous to the above. When they affect patients with heart disease and much weakness of the heart's action, the latter may constitute a fatal obstacle to the removal of the secondary disturbances of the cerebral circulation brought about by the encephalitis. If the disease of the heart is recent, and the compensatory hypertrophy is sufficient, the disturbances are easily overcome.

c. Tumor.—A great number of the symptoms which are observed in tumors of the brain must be ascribed to the secondary encephalitis in the surrounding tissue, with its general action on the cerebral circulation. Since a large number of the tumors are located in parts of the brain where the symptoms of local disease are not produced at all, or appear only at a later period, when the tumor has increased in size and involves neighboring parts, the initial signs will be altogether general in a great majority of cases (cephalalgia, diminution of the psychical powers, disturbances of a general nature in the organs of sense, and the like). Even these "general" disturbances are also modified most essentially by the accompanying changes in the surrounding parts: intercurrent fainting-fits, attacks of vertigo, partial and general convulsions, obscure febrile conditions, transitory stupor and somnolence—symptoms which point to a variable element accompanying the tumor, and are partially at least the results of inflammatory reaction and its consequences. Even those tumors which are so situated as to cause at once symptoms of localized disease (paralyses of motion and sensibility, etc.), are very greatly influenced in their course by the changes of the surrounding brain-substance. We believe that we must ascribe to more or less wide-spread encephalitic disturbances around the tumors a large proportion of the sudden aggravations of hemiparesis to hemiplegia, or of the sudden apoplectiform attacks of hemiplegia, many of the intercurrent convulsions of a partial character with subsequent increase of the paralysis, many general convulsions, and many sudden cases of death preceded by rapid extinction of the functions of the brain. Unfortunately, however, it is as yet impossible to pick out with certainty from these manifold symptoms those which are to be ascribed to the encephalitis; for in the essence and growth of many tumors we find a factor which is quite as variable as the encephalitic processes, viz., the degree of the vascular distention. The tumor can at times cause great compression of the brain without any accompanying encephalitic swelling, while its injurious influence is at other times slight. In some few cases where we expected to find wide-spread encephalitis of the parts surrounding the tumor, it was not present, and all symptoms of irritation had to be ascribed to the tumor itself.

Psychical disturbances occur in about one-third of the cases of cerebral tumor (Ladame); this is not surprising when we remember that a very large number of tumors during a great part of their course do not injure the cortex of the cerebrum at all, or do so only very indirectly; the tumors of the base and of the cerebellum, especially, do not involve the cortex, or at least they do not involve it for a long time. If, however, only the last stages be taken into consideration, the statistical proportion is entirely different. It is instructive to see in connection with tumors which lie near the cortex of the cerebrum, how this cortex, which macroscopically appears to be normal, presents a number of changes entirely similar in nature to those already briefly described while speaking of the encephalitis around apoplectic foci. We are forced to admit that the transitory congestive conditions, which, accompanied by exacerbations of the headache, are so characteristic of tumors, are the principal cause of the temporary retardation of the circulation in the cortex, not alone in the immediate neighborhood of the tumor, but also in widely extended portions. The ultimate effect is atrophy of the cortex. Here the direct irritation produced by the growing—and, when it is very rich in vessels, at times congested—tumor may play an important rôle as a causative factor, especially when we have to deal simply with signs of psychical torpor. When, however, active signs of irritation occur, we cannot be too ready to suspect an accompanying red softening; and often enough the autopsy confirms the diagnosis. It is exceedingly difficult to say whether the epileptiform convulsions are caused by the encephalitis and the cerebral compression excited by it, or by an enlargement of the tumor itself, and the consequent anæmia of the medulla oblongata. The decision can generally only be made at the autopsy, and sometimes not even then.

From the fact that oftentimes a series of convulsions occur for weeks and then disappear for a long time, we can draw no definite conclusion as to the existence of a complicating encephalitis, for an absence of inflammation, for example, in tumors of the convexity, has several times been proved. The convulsions that are confined to special groups of muscles afford stronger proof of an accompanying encephalitis, especially those limited twitchings which are followed by paralysis of the affected muscles. In such cases it advances from the circumference of the tumor somewhat rapidly into the tissue of the brain, causing an active irritation of the fibres before it destroys them; in this connection, it is true, many essential pathologico-anatomical and physiological points are still obscure. We are very insufficiently informed concerning the degree of irritability of the fibres of the corona radiata; we have no knowledge at all about the conditions of the fibres in which they no longer act as conductors, but are capable of resuming their functions; further, we are pathologico-anatomically ignorant of the causes (and the point of attack of the irritation) of convulsions in paralyzed limbs. We have, however, in spite of our very meagre knowledge, derived the conviction from a series of autopsies, that the motor disturbances of the last category are almost without exception to be ascribed to the accompanying encephalitis. The same applies to the contractures, which, it is true, are observed much more rarely than the convulsions; there is no unanimity in the explanation of the contractures, authors having divided themselves into two parties: one ascribes the contractures to the accompanying encephalitis; the other regards them as the result of the development of the tumor. Our experience indicates that both views are correct. Among the paralyses there is one which can, with tolerable certainty, be ascribed to the accompanying encephalitis or apoplexy. There are the sudden "apoplectiform" paralyses. If they are preceded by a short stage of rapidly increasing symptoms of compression of the brain, to which the paralysis was suddenly added, they are probably due to encephalitis; if an initiatory stage is wanting, the cause will more likely be found in an apoplexy near the tumor. When the paralysis is developed very slowly, it must be ascribed to the enlargement of the tumor. These paralyses are mostly hemiplegias. Ladame finds them in about one-third of all the cases.

The disturbances of sensibility which accompany the tumor may also be caused by the encephalitis, or, if already present, can be essentially influenced by it. Every secondary encephalitis, be it ever so slight, causes a violent headache; all the attacks of severe headache must not, however, be ascribed to encephalitis, for they may just as well be due to a congestive swelling of the tumor. Every encephalitis around the tumor which involves the sensitive fibres of the corona radiata is liable to cause disturbances of sensation in the opposite side; but, for the reason given above, every such disturbance of sensation should not be ascribed to a complicating encephalitis. In certain cases, therefore, a decision is often impossible.

Patients with tumors of the brain frequently fall into a transitory state of sopor, which sometimes leads to rapid death. The opinion has already been expressed that in these cases we always have to deal with encephalitic processes. This is not correct. It is unquestionably possible for an encephalitis to involve a great extent of the cerebral tissue around the tumor; it may then cause considerable pressure on the brain, and finally cerebral œdema which destroys life. But other cases terminate in the same way, and we find that it was the tumor itself which produced the pressure and the final interruption of the circulation.

These few remarks will suffice to indicate the connection of encephalitis with tumors of the brain. To enable us to understand this connection perfectly, further experimental investigations of the excitabilty of the fibres of the corona radiata under different circumstances are necessary, as well as a much more accurate pathologico-anatomical study of cerebral tumors.

IV.—Chronic Abscess of the Brain.

I. *Traumatic abscess.*—Acute encephalitis after injury has been briefly described above; among the terminations of the same, it was mentioned that death might occur in the period of red softening, and attention was specially called to acute suppuration of the brain, which leads with comparative rapidity to death. But it was stated, at the same time, that all, or nearly all, the cerebral symptoms may disappear and a relatively free period, which we have designated *as the latent stage of chronic abscess of the brain*, may follow.

There must be anatomical conditions, of which we are as yet unable to gain an accurate knowledge, and which in general consist in this, that the congestive swelling subsides and the organ regains its normal size. It will now depend on the size and locality of the focus, which very probably already contains pus, whether the latent stage takes the form of a real intermission, or whether cerebral symptoms appear from time to time during it.

Experience has shown that there are pure and impure latent stages, and the periodic symptoms of the latter are of great weight in the diagnosis of abscess. Small abscesses, of course, will be longer borne, although they are quite as capable as the larger ones of causing the usual symptoms when they occupy favorable places (nucleus lentiformis, corpus striatum, capsula interna, left insula and its neighborhood, etc.). When, however, all those portions of brain which, according to our experience, furnish symptoms of local disease when injured, remain unaffected by a latent abscess, and when only co-ordination systems in the white substance of the hemisphere, or superficial portions—unimportant in a motor and sensitive relation—are injured, distinct symptoms of local disease will not be developed. An accurate investigation of the psychical functions discovers a psychical defect, a weakness of memory as well as of rapidity and readiness of thinking and judging under such circumstances; but it can occur to no man to draw therefrom a conclusion that a certain portion of the cortex of the cerebrum has been destroyed. From this it will be understood that we find symptoms of local disease in rather more than half of the cases of suppuration of the hemispheres, while the others run their course with only diffuse disturbances and a few very striking episodes (convulsions, etc.); and even these, also may in rare cases be wanting.

Duration of the latent stage.—All authors mention the extraordinary differences in the length of this stage. Lebert estimates the duration at from one to two months, and, as it appears to us, has hit upon a tolerably accurate average. It is known, however, that very short latencies of a few days' length occur, while a few cases where the latency lasted twenty years and more can scarcely be doubted. We give a few examples which will serve to illustrate this:

Eight days (our own observation), fourteen days (Hofmann), six weeks (Chinault), eight weeks (Riecke), seventy days (Adelmann), three months (Schroeder van der Kolk), three and one-half months (M'Naughton), four months (Downs), five months (Griesinger), six months (Groell), twelve months (our own observation), six years (Sander), nine years (Stueve), twenty-one years (Gerhardt and Schott), twenty-six years (Haerlin). There have also been recorded a great number of abscesses of the brain where the latency varied within the boundaries mentioned.

If, now, the latent period is very short—a few days—and is followed by one of acute brain-symptoms, which in some cases may be greatly prolonged, the characteristic course of abscess is entirely obliterated, and transition forms between acute and chronic abscess of the brain are seen which cannot be reckoned among chronic abscesses, because, in spite of their frequently long duration, they do not present the typical course of such. In such cases recognition is often impossible.

Symptoms of the Latent Stage.

a. Latencies of absolute completeness have been seen during which not a single symptom called attention to an intra-cranial affection (Bianchi and Merkie, Schroeder van der Kolk). Such cases are very rare; for, even if symptoms of localized disease and the like are wanting, a chronic, occasionally exacerbating headache, is rarely absent. There appear, indeed, to be circumstances where the anatomical relations are such that the abscess exercises no pressure on the surrounding parts, but merely plays the rôle of a loss of substance in the brain-tissue, on the locality of which it depends whether any symptoms at all occur or not.

b. Symptoms of localized disease during the latent stage.—As soon as the primary lesion affects a spot in the brain which possesses a definite value with regard to distinct functions, the special results of the lesion of this portion of the brain will continue in the latent stage during the transformation of the initial lesion into an abscess. In many cases this is characterized by symptoms of localized disease, which under certain circumstances allow a tolerably sure opinion to be formed concerning the locality of the focus. These signs are the following:

1. *Aphasia.*—Boinet describes a very instructive case, where, after an injury (blow on the left side of the forehead), trephining was performed, and aphasia remained, although the patient was otherwise perfectly healthy and intelligent. A long time afterward he died suddenly in an epileptic attack. The autopsy showed a sharply circumscribed abscess of Broca's convolution (first frontal convolution) reaching to the corpus striatum. At the same time the patient had paralysis of the right facialis during the whole time, a fact of no less interest.

Here, too, the fact is usually confirmed that so soon as a lesion of the immediate neighborhood of the cortex of the left island of Reil occurs, especially the convolutions directly above it, aphasia takes place.

2. *Hemiplegia during the latent stage.*—Wyss describes an

abscess of the brain in a boy ten months old, in which the stage of latency, not perfect certainly, was marked by paralysis of the right side. We have seen a case ourselves, a report of which we cannot give, where a ten weeks' latency was characterized by a paresis of the left side and of the left facialis; no aphasia. The abscess occupied the left frontal lobe, and had almost entirely destroyed the first convolution. This observation forms a set-off to the one of Boinet above given, where an analogous affection of the left side led to aphasia.

3. Convulsions limited to a few groups of muscles during the period of latency are very rare. The convulsions of this period are very much oftener general. Not very unfrequently the convulsions begin in certain groups of muscles (facialis, arm, leg, or the arm or facialis is not affected); at the end of the attack we notice sometimes that the contraction leaves certain groups of muscles last. We consider this a point of some value in the diagnosis of a focus, from which as a centre the convulsions are excited in a manner not accurately known. With reference to this last we are, in fact, thrown back on pure hypothesis.

4. *Strabismus.*—Very rare, and noted in only a very few cases.

c. Signs of a constant, moderate pressure on the brain during the latency.—In spite of an unaffected sensorium and a normal psychical capacity, a constant headache during latency has been seen by a few observers; though latent abscesses show a *constant* cephalalgia only in a small number of cases. Then the patient is in fact never free from pain; exacerbations occur, and now and then light febrile movements also. Dizziness, nausea, and occasional vomiting usually accompany the exacerbations of pain. The vomiting appears to occur pretty often during the latency, and has not always been properly appreciated. This headache may be confined to the spot where the injury was received, or the exacerbations at least may begin there; in other cases it corresponds to that portion of the brain in which the abscess develops, which is by no means always at the place of the primary injury. Violent exacerbations of headache indicate in most cases congestion around the abscess; the more frequent these attacks, the more must one be prepared for the occurrence of changes around the abscess, which may quickly lead to death (acute red inflammatory softening, yellow softening, both with extensive œdema of the brain). Therefore acute attacks of headache occasionally indicate the commencement of the terminal period. Constant headache in abscess of the brain is under all circumstances to be referred to tension of the contents of the capsule; the size of the abscess, the acuteness of its formation, and the promptness of the occurrence of the terminal processes, have much less to do with it. Tension of the capsule is dependent on influences the nature of which we are still unable to discover.

Occasionally other general symptoms accompany the chronic headache, which confirm the explanation of the pain by pressure: change of behavior, other reactions, especially dulling of the senses, slowness, want of motor energy, a gradual imbecility, may accompany the course of the cephalalgia (Silferberg, Gull). Occasionally these disturbances assume the form of a somewhat more distinct set of symptoms; we have seen a patient who was periodically afflicted with manifold optical illusions; for a time he was subject to intermittent attacks of extreme terror. The patient was an interesting example of melancholia following a blow upon the head. After a latent period of nine months, an acute terminal stage with convulsions put an end to the psychosis (abscess in the left occipital lobe, without any symptoms of localized disease). We have never seen any mention of violent delirium and maniacal excitement under the above circumstances in the latent stage. The sleeplessness mentioned by a few observers may also be placed in this list of symptoms. The constant headache is without doubt to be attributed to the dura mater. In the above-mentioned patient a moderate congestion in the retina of both eyes was found during life; at the autopsy both optici showed an ampulla, which, however, must be ascribed to the great pressure on the brain during the terminal stage.

d. Signs of great intermittent pressure on the brain, with intervals of relief. It may happen that the patient, previously free from all head-symptoms, soon becomes somnolent after rapid development of a severe headache, and falls into a deep but transitory coma of several hours' duration, accompanied by febrile movements (Hayden). Hayden's patient died in such an attack of coma; the abscess was located in the left thalamus. Without accurate results of post-mortem examinations, especially without accurate examination of the circumference of the abscess, the cause of the attacks of coma cannot be given with certainty; the most probable explanation is that they are due to congestive pressure on the brain, either excited by the abscess itself or by encephalitis of the surrounding parts.

e. Signs of a collection of pus which is not entirely shut off from the vascular system. Chills have been observed not very unfrequently during the latent stage (see Kistel, mentioned in Bruns, p. 966, Oedmansson). Two cases occurred to us, which, in consequence of our total ignorance of the etiological factors, and the entire coincidence of the symptoms with intermittent fever, were regarded as such. In the first case there were three chills of the regular tertiary type, followed by heat and sweating; during the attack a very considerable swelling of the spleen and liver occurred, which diminished, but did not entirely disappear in the apyretic stage; no brain-symptoms except slight headache. The patient did not recover from the second attack, but went into a sopor and coma, and died after a few hours. The autopsy showed a great abscess in the right occipital and temporal lobes. The second case is as follows:

39. An Italian railroad laborer, twenty-four years old; admitted the 4th of November, died the 23d of November. In summer of 1872, working as a bricklayer in Trieste, had intermittent fever for the first time; three attacks during August, September, and October; entered hospital for treatment. From November, 1872, to October, 1875, lived in Turin, where he was perfectly well and free from intermittent fever. A month ago came to Switzerland, and on October 27th had fever again for the first time. Patient gives no history of injury.

November 5th.—He is a strong individual; no nervous symptoms; chest organs entirely normal, likewise abdominal organs, with the exception of a moderate swelling of the spleen. Urine normal. On November 4th, at three o'clock P.M., chilly sensations; temperature 40.6° (105.08° F.); after four o'clock, sweating; six o'clock in the morning, temperature 38.6° (101.48° F.). On November 5th, four o'clock in the afternoon, chills again; temperature 39.6°–40° (103.28° F.); at six o'clock, 39.8° (103.64° F.); sweating.

November 6th.—Free from fever; three in the afternoon, 37.4° (99.32° F.); ten o'clock in the evening, 37.2° (98.96° F.). On the evening of November 6th, enlargement of the spleen, and a considerable increase in size of the liver discovered.

November 7th.—Free from fever; subjective condition good; no head-symptoms; spleen swollen. Since yesterday the liver much decreased in size, its posterior boundary of dullness considerably lower. 37.4°–38.8° (99.32°–101.84° F.).

November 8th.—Free from fever; no regular attack of intermittent yesterday; no chill; no separate stages. To-day the liver is of normal size, not swollen; the spleen likewise smaller, approaching the normal.

November 9th.—Yesterday afternoon, up to five o'clock, regular increase of temperature to 39.2° (102.56° F.); since, reduction of the fever to 38.6° (101.48° F.), but no true defervescence. Liver shows no change in size to-day, yet the spleen swells up again. Yesterday increase of the fever to 39.6° (103.28° F.); to-day no total falling off.

November 10th.—Yesterday the old course of fever, without chill; subjective condition the same; no head-symptoms; no complaint of pain; spleen swollen; liver remains small.

November 11th.—Yesterday, increase until afternoon, 39.4° (102.92° F.); then decrease until this morning. No new symptoms. Energetic treatment with quinine.

November 12th.—Free from fever all day yesterday.

November 13th.—Free from fever. Spleen still large. General condition good.

November 14th.—Free from fever. Vomiting twice from quinine. Spleen swollen.

November 15th.—Free from fever. Vomiting, which is attributed to quinine. General condition somewhat altered, yet no head-symptoms, except complaint of headache.

November 16th.—Complete apyrexia; complaint of headache; no vomiting, but disturbed general condition; spleen swollen.

November 17th.—Free from fever. No more vomiting; loss of appetite; headache; constipation.

November 18th.—Free from fever. Headache and constipation; yet no other nervous symptom; no more vomiting.

November 19th.—For the last few days patient has had, with low temperature, a remarkably slow pulse, 48. Free from fever. One stool; no vomiting; moderate headache; sensorium entirely normal, but patient looks lean and shrunken.

November 20th.—Free from fever. Pulse 48 to 52. Other symptoms quite the same.

November 21st.—For the past few days the patient, without showing distinct head-symptoms, has been very quiet and melancholy; free from fever. Pulse 44 to 56. Headache. Has had one stool; no vomiting. Sensorium entirely normal. To-day the morose, peculiar condition is more marked; he gives few answers; wraps himself in the clothes. Turns away, which is attributed to ill-nature.

November 22d.—Quiet night; absolute freedom from fever. Pulse 60 to 72. Quiet sleep; lies quietly in bed; resists attempts to have it made up; will not allow himself to be moved; makes all sorts of irrational repelling movements; stretches and rolls himself in bed; does not answer; is not in his senses; pushes away all drink, and the ice-bladder also. All questions about pain and wants are answered with a constant "nothing." Urinates in his bed. Meanwhile he is free from fever. Pulse 80, and small. Dilatation and feeble reaction of both pupils; reflex irritability on touching the cornea diminished; no strabismus; left cheek somewhat more flabby than the right. Tongue is not protruded. No delirium; quiet posture, but tries to repel any interference. No rigidity of neck; no vomiting; abdomen somewhat sunken; lungs and heart normal; dullness of spleen somewhat enlarged. About two hours later somewhat better, but extraordinarily indifferent; does not show the slightest interest at a visit, and when not spoken to does not say a word. Temperature 36.6° (97.88° F.). Pulse 80. Temperature 38°, and pulse 80 the whole day unchanged. Urinates in bed. Pupils remain equal.

November 23d.—Temperature 38° (100.4° F.). Pulse 120. Temperature 38.2° (100.76° F.). Pulse 96. Out of his mind; sopor; left eyelid less closed than the right; ptosis of the right. Left pupil moderately large; right pupil considerably larger; partial paralysis of the oculo-motorius on the right side. Mouth open; cheeks are blown out with expiration. Both angles of the mouth equal; reflex movements not excited by severe irritation of the face, and remarkably slight even on irritation of the cornea. Abdomen sunken like the hollow of a canoe; no spontaneous movements, and reflex excitability greatly diminished everywhere. No convulsions. Patient's condition such that the degree of sensibility cannot be tested. Slight increase of fever this morning; pulse all at once 120, small; diminishes in frequency, however, during the day again, and becomes fuller. Dullness of the spleen somewhat increased.

At 12 o'clock, temperature 37.2° (98.96° F.). Pulse 116. At 12.37 o'clock, sudden change of the color of the countenance; paleness, followed by lividity; death, without any further symptoms involving the lungs or nervous system.

Autopsy.—Abscess in the left frontal lobe in the substance of the hemisphere proper, outside the caudex cerebri.[1] Abscess in the right temporal lobe underneath the pes hippocampi major. Ampullæ in both optici. Bronchial glands cheesy; slight melanosis of the spleen: slight degree of melanotic change in the liver. (Our own observation.)

This case shows that abscess of the brain may be mistaken for intermittent fever, especially for one of irregular form, and the error is hardly to be avoided. The case is also of value for its terminal stage and its very peculiar course.

e. Convulsions.—Epileptiform convulsions have often been seen during the latent stage and have been regarded as true epilepsy (Hutchinson and Jackson). This cannot cause wonder when one remembers the total lack of symptoms of abscess of the brain in certain cases, and the absolute similarity of certain epileptic attacks to true epilepsy. It is therefore well, especially when the epilepsy dates back to an injury, fall, or blow, to keep in mind the possibility of an abscess of the brain. In some cases this abscess-epilepsy does not differ from true epileptic convulsions in a single symptom. It is always a rare occurrence in the latent stage, while in the terminal one, where we shall meet with it again, it is more frequent.

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[1] Stammfaserung: this refers to the cerebral ganglia, together with the crura cerebri, which are intimately associated with them.

Its cause, under these circumstances, cannot be determined; it certainly has nothing to do with a slowly increasing pressure on the brain, caused perhaps by its swelling at the end, or in the course of which epileptic convulsions occur; but very suddenly, as in an ordinary seizure, the patients fall and the attack runs its course. In such a case there can be no question of a gradually produced anæmia of the medulla oblongata.

The behavior of the abscess during the latent stage, as has been already intimated, is very peculiar and varies in the different cases:

1. According to its position it causes symptoms of localized disease, or remains entirely latent.

2. In some cases the focus makes pressure; in others it makes none at all, and acts only by removing a part of the brain-substance.

3. In some cases it leads to no congestive processes, so that no intercurrent brain-symptoms occur; in others this is a daily occurrence.

4. In some cases it leaves the surrounding brain-substance entirely intact; in others it may result in red softening or yellow necrosis of the surrounding parts to a great distance. In this way it can increase in size.

From this it appears that a number of essential qualities and processes, which play a part in abscess of the brain, must be yet unknown to us.

The terminal stage.—One is generally inclined to consider the terminal processes as connected with the rapid growth and increase of the abscess, and in this a mistake could scarcely be made; but it must be remembered that the growth hardly represents a new function suddenly acquired by the abscess, but that previously, during the latent stage, signs occur often enough which indicate a temporary increase in size of the entire formation, which then, after causing more or less severe reaction, remains constant and is afterwards supported. The terminal stage of abscess of the brain is more or less distinctly a sequence of the latent one; it leads, in the great majority of cases, to an early death; only a small number show a recovery from attacks which resemble the terminal processes, so that a kind of second latency results, which, however, is not of long duration. It is a fact which was proved statistically by Rudolf Meyer, that when once the terminal period begins, the greater number of abscesses of the brain lead to death in the first week. (Of thirty-four abscesses of the brain in which it was possible to accurately determine the duration of the terminal stage, thirteen ran their course within one week, seven in two, four each in three, four and five weeks, one within six weeks, and one within nine weeks).

The terminal processes are:

1. Perforation outward on the surface, and consecutive meningitis of the convexity.
2. Perforation at the base and consecutive meningitis of the base.
3. Perforation of the pus into the ventricles.
4. Extensive œdema of the brain, due simply to the enlarging abscess.
5. Œdema of the brain, consequent upon fresh encephalitis.
6. Compression of the medulla oblongata in abscess of the cerebellum.
7. Occasionally the brain is found in a condition of remarkable anæmia without œdema; the more immediate causes which have produced death here are not clear.

The above mentioned are not normal terminations of all cases; on the contrary, they must be regarded as exceptions. The usual termination is the one given under No. 4; the abscess commences to increase under unknown influences, its contents increase considerably in amount, and exert a symmetrical pressure in all directions, which sooner or later paralyzes the circulation in the entire brain. The universally increased pressure obstructs the circulation by compression of those vessels which are most compressible—the capillaries; active congestion follows, but cannot remove the obstacle. Considerable increase of arterial pressure, transudation throughout the entire brain, and diminution of venous pressure result. Under such circumstances œdema of the brain is not to be wondered at; this finally forms the chief obstacle to the influx of fresh blood, and under such circumstances the brain must, according to known laws, discontinue its function. In such a process there are no grounds for symptoms of local disease—in fact, in a great number of cases they are entirely wanting; such are those in which the focus is situated apart from every sensitive and motor fibre of the corona radiata. In the opposite case, however, the focus reaches these regions or the irritable portions of the cortex by its growth; very violent symptoms of local disease usually result.

Furthermore, the circumstance whether the latent stage was or was not free from symptoms of local disease, will have great influence upon the nature of the terminal stage.

When the terminal œdema of the brain depends on complicating fresh encephalitis, then the symptoms cannot be differentiated from those of the increasing abscess. The latter causes symptoms which can scarcely be distinguished from fresh encephalitis.

Meningitis and perforation into the ventricle can be recognized in certain cases, but by no means always.

a. Terminal œdema of the brain.—We have already briefly expressed our opinion of its origin. We are entirely in the dark as to the immediate cause of the sudden increase of the abscess after it has been at rest for a length of time.

Before the terminal signs appear, the physician has either had reason to suspect an abscess (as in cases with history of an injury, or those in which the latent stage was not entirely free from symptoms, but showed those above mentioned), or has never thought of any lesion of the brain (see the above-mentioned case), because the etiology remained unknown; or the patients were regarded as epileptic, or as affected with intermittent fever, or as dull of intellect; and he thinks of atrophy of the brain due to injury, or of chronic meningitis. Now a new series of symptoms, leading quickly to death, appear:

A pressure on the brain is quickly developed (from a few hours to a few days), with all the characteristic appearances, which in the majority of cases soon leads to death, and allows no variations to be recognized; yet cases have also been seen where the affair ran its course with frequent improvements and aggravations, and finally led to death. The commencing pressure on the brain frequently makes an initial stage of irritation, which, however, quickly passes by and usually escapes observation. We find symptoms of psychical irritation, restlessness, illusions, violent delirium, associated with a rise of temperature which is seldom very great, unless, as in the above described case, the remains of a preceding periodic fever should be mingled with it. An unsettled and torpid condition of the sensorium follows very soon upon the delirium—soporific or half-soporific conditions, mostly not admitting of a sure interpretation, and very differently comprehended by observers (melancholia, simulation, and the like). In the meantime the patient commences to complain of very severe headache; his mind grows less and less clear; his temperature falls (febrile conditions are the exception in such cases); his pulse becomes slow; his pupils wide, and react feebly; his retina shows passive congestion; in some cases vomiting occurs; the sopor increases to coma, with entire loss of reaction; absence of all excitation originating in the brain; disappearance of all brain and spinal reflex actions. The evacuations are involuntary. The pulse remains slow; this condition may last for a few days, more or less, and then the pulse becomes quick, small, irregular; the temperature shows isolated variations, which now and then reach the fever line, and in which no law is apparent. The centres of the medulla oblongata succumb, without other noticeable symptoms, to the increasing pressure in the skull, after those of the cerebrum and spinal cord have long since lost their activity (absolute extinction of reflex action). The terminal stage of those abscesses which do not reach the surface of the brain, and which involve the terminations of no important set of motor and sensory fibres, runs its course thus with slight variations. We have seen this kind of death several times, especially in abscesses in the temporal lobe. But convulsions are not always absent in the terminal stage, when the abscess occupies this position ; they can occur even when the cortex and the surface of the ventricles are not reached by the abscess. They may be partial or general ; in the first case they leave, not very unfrequently, hemiplegia behind ; but paralysis can follow general convulsions also. A general epileptiform convulsion in the terminal stage, so long as only œdema of the brain is present, is rare, and its origin is not to be given with physiological certainty. It is followed by coma, if this is not already present. Partial convulsions must be connected with the irritation of the system of fibres which run by the abscess. They are also occasionally disabled at the same time, for paralysis follows ; a positive experimental basis, however, for this opinion, is still wanting, for the well established fact that convulsions occur, although very rarely, in limbs that have long been paralyzed, seems to show that other modifications of irritation also exist. Till these deficiencies are filled out, convulsive symptoms must be attributed, in general, to the growth of the cavity of the abscess.

Any symptoms of local disease that were present in the latent stage are variously modified in the terminal stage. Partial convulsions which were present become more violent, and lead to paralysis, or they undergo a transformation into a series of general epileptiform attacks. Paralyzed limbs suddenly begin to twitch. It is to be remarked that hemiplegia is certainly the most frequent form of paralysis, but that also isolated paralysis of the facialis, of an arm, a leg, of the face and arm of the same side has been seen, and the inexactness of the account of the autopsy in those cases is greatly to be regretted. One case (Holzhausen) showed paralysis of an arm and both legs. Pure paraplegia has not been seen in abscess of the brain.

Contractures occur likewise in the terminal stage, without perforation on the surface of the brain or internally having taken place. Whether they depend on fresh encephalitic processes or not, we are not able to determine. Isolated very remarkable cases are to be rated as exceptional cases, and certainly, in part at least, are to be regarded as incomplete observations. Thus, for example, the case of Andral, which stands alone in literature, where with suppuration of the glandula pituitaria the entire body was permanently contracted toward the right. R. Meyer has collected (Diss., p. 54) a number of cases in which the locality indicated by the symptoms of local disease did not accord with the lesion present in the hemisphere. With regard to this we have become extremely skeptical ever since; in two cases where paresis on the same side with the apoplectic spot caused astonishment, we found the cause in foci of the other hemisphere, which had been overlooked.

Sensibility.—In a process like the one under discussion, the chief symptom of which is an alteration of the consciousness, many and important disturbances of sensibility escape observation. Cephalalgia, in connection with dizziness, delirium, and vomiting in different combinations and intensity, is the chief thing. Distinct changes in the sensibility of the surface of the body in the terminal stage are occasionally, although very seldom, seen. R. Meyer gives as a result of his statistics that only in seven cases out of ninety is mention made of formication, dysæsthesiæ, as present in the limbs; twice only of anæsthesia dolorosa in the extremities; four times of neuralgia of the trigeminus; twice the neuralgia was upon the same side as the focus. These data in these statistics relate to all abscesses of the brain collectively, therefore the number of the disturbances of sensibility in the terminal stage will be still smaller.

b. Combination of meningitis of the base or convexity, with breaking of the abscess outward.

Abscesses in combination with meningitis are recorded, in which the latter did not cause death immediately, while in other cases the course is an eminently acute one, limited to a few hours. The reasons for these differences are not clear in single cases. We shall also find that the duration of the case varies after perforation has taken place inward.

40. A soldier, twenty years of age. December 10th, 1870, received a grazing shot on the right side of the head; December 14th, admitted to hospital.

December 19th.—Superficial sloughing of the skin on the right side of the head where the bullet had passed.

January 10th.—Surface of the wound larger, dirty gray.

January 15th.—Enlargement of the wound, a circular hole of four centimetres; dirty gray appearance. Liq. ferri sesquichl. Lower angle of the wound five centimetres (two inches) above the meatus auditorius externus; bone laid bare for length of three centimetres (one and one-half inch).

February 4th.—Wound looks somewhat worse; on the front edge a whitish deposit.

Headache for the first time; sudden attack of convulsions in the region of left facial (mouth, nose, orbicularis palpebrarum). The muscles of the tongue and of inspiration also shared in the attack, to this extent that hiccough synchronous with the other convulsions took place; also the right sterno-cleido-mastoid. Color of the face white as chalk; five minutes' duration.

Temporary paralysis of the whole of the left facial and the muscles of the left half of the tongue followed; after a few minutes it abated, first in the upper branches of the facial; no loss of consciousness during the whole attack.

Ten minutes afterwards analogous contractions in all the flexors of the left hand.

The normal color of the countenance is again present; no change in the pupils. Pulse strikingly accelerated, right one much smaller than the left. The arteria radialis less tense; after the attack, vice versa.

Midday, a similar attack.

Evening, temperature 39.8° (103.64° Fahr.). Pulse 120.

February 5th.—Vomiting. It is evident that the mind is slightly affected, for the entire train of thought turns on a lack of appetite. Temperature 39.3° (102.74° Fahr.), pulse 120—39.9° (103.82° Fahr.), pulse 120.

February 6th.—Temperature 38.9° (102.02° Fahr.), pulse 100—39.8° (103.64° Fahr.), pulse 120.

February 7th.—Range of thought confined, yet no delirium. Slight paralysis of the left side of face. Slight spasm of the depressor anguli oris, orbicularis oris, and the muscles of the left side of the nose, occurring every half-minute.

Tongue deviates to the left, uvula to the right; sensibility everywhere intact. Temperature 38.3 (100.94° Fahr.). Pulse 100.

At three o'clock in the afternoon a new attack in the region of the left facialis; the left abducens and right rectus internus also take part in it. At half-past three a new attack, with participation of the pectorales and the muscles of the abdomen.

Shortly afterwards a repeated attack, during which it is ascertained that the patient can voluntarily move the right hand, that the contractions are confined to the right facialis, the right sterno-cleido-mastoideus, and all the muscles on both sides between the lower jaw and sternum. The patient understands and remembers what is said to him.

Evening temperature 39° (102.2° Fahr.). Pulse 100.

February 8th.—Attack in the morning; duration an hour, without loss of consciousness; convulsions in the left arm and in the left half of the thorax; at the height of the attack, also in the right arm. Temperature 39.6° (103.28° Fahr.), pulse 110—40.5° (104.9° Fahr.), pulse 120.

February 9th.—Temperature 39.3° (102.74° Fahr.), pulse 110—39.6° (103.28° Fahr.), pulse 120. In the evening, snoring, stupor, and another attack.

February 10th.—Sopor, snoring; right pulse smaller than the left; left pupil strongly contracted, without reaction; right reacts a little. Abdomen not boat-shaped. Temperature 38.4° (101.12° Fahr.), pulse 120—41.8° (107.22° Fahr.); post-mortem rise to 42° (107.6° Fahr.).

Autopsy.—On the external table a distinct line of demarcation was found of conoidal shape; at the corresponding point on the inner table the bone covered with thick yellow pus, gray, and discolored over a surface as large as a gulden (twenty-five cent piece). A splinter of bone, the size of a lentil, chipped off and only loosely attached to the vitreous table. Half a spoonful of greenish pus escaped from a corresponding hole in the dura mater; the entire dura of the right convexity covered with yellow pus on its inner surface. Pia mater of the right convexity, from front to back, covered with thick pus, and transformed into a thick membrane; in the neighborhood of the abscess of the brain, fluid pus. Abscess on the surface of the brain from one and one-half to two centimetres deep; external opening of the same size. Upper edge six and one-half centimetres from the median line, posterior part two and one-half centimetres in front of the middle part of the fossa of Sylvius, immediately on the front border of the fossa Rolandi, between the latter and the gyrus præcentralis.

The cortical substance about the abscess very soft (Hitzig).

This case is one of the choicest examples of the localization of definite motor functions in definite parts of the cortex. The centre of innervation must be sought there (paralysis of the facialis and hypoglossus of the other side), where, through a process which also aroused convulsions, a definite portion of the surface of the brain perished. The time of the occurrence of terminal meningitis is not to be determined with all the necessary certainty, yet no doubt can be entertained of its commencement a few days before death, on account of its anatomical character.

Most observations of meningitis of the convexity with perforation, however, showed a much quicker course. The symptoms are those of an extraordinarily intense irritation of the brain, with convulsive symptoms—very seldom partial, usually general in nature—with delirium, prompt loss of consciousness, and early coma. It is superfluous to again mention here the symptoms of meningitis of the convexity running a rapid course.

c. Perforation of the abscess into the ventricle.—This occurrence causes a train of symptoms which can be recognized under certain circumstances. There are, to be sure, no pathognomonic symptoms, but a combination which has repeatedly occurred in many cases.

41. Biermer (communicated in Meyer, Diss.): An abscess of the left hemisphere breaks through into the ventricle, and this occurrence puts an end to a pure latency. Chills are mentioned, which probably were convulsions. After a few days, paresis of the right arm, then rapid paresis of the lower extremities. Abolition of consciousness; in the meantime convulsions and frequent vomiting.

42. Meyer: Bilateral convulsions of the face, with delirium and loss of consciousness, which again improved. Afterwards increased headache; delirium; death.

43. Our own observation: Breaking through of an old necrotic focus (in which a fresh hemorrhage had taken place) into the ventricle; consciousness already previously disturbed, and left hemiplegia. Stiffness of the entire healthy side; twitching of the muscles of both eyes, followed promptly by death.

44. Our own observation: Breaking through of a large necrotic focus of softening into the ventricle. Convulsions in both legs, with preservation of consciousness for twenty-four hours; then convulsions in the muscles of the eyes, delirium, coma, death with the usual symptoms of meningitis.

To these few examples it would be easy to add a number of others. It is readily perceived that the recognition of this incident is only possible when the abscess or other focus has been previously diagnosticated, which, according to what has been said, will not always be the case in abscess. If the abscess is surely diagnosticated, the possibility of this occurrence must always be kept in mind. A sudden cerebral attack, which indicates with certainty a simultaneously occurring irritation of motor centres of both hemispheres (convulsions of both legs, of both faciales), is the surest point when the intellect has previously been intact. This integrity of the sensorium, however, lasts, according to our own observations, only a short time; then clonic spasms of the muscles of the eyes appear (diffusion of the inflammation of the plexus posteriorly to the region of the corpora quadrigemina), followed very quickly by delirium and profound alteration of the intellect (dissemination of the inflammation over the pia). At the same time paralyses of the extremities usually occur, of which hemiplegiæ are the most frequent.

The extent of the convulsions in the initial stage may be considerable; general convulsions have been seen. The course of the affection, as thus briefly sketched, is not uncomplicated in all cases, especially when disturbances of consciousness and convulsions have been present; if, however, it comes under observation under otherwise favorable conditions, the diagnosis can be ventured.

The period of survival after perforation into the ventricle is, under all circumstances, short; from four to twenty-four hours are the extreme limits which are known to us.

We have now described the course of acute and chronic abscess; in regard to the latter, we have specially had in view typical cases, in which something can be accomplished in the way of diagnosis. Besides, abscesses of the brain are occasionally met with,—the records show a whole series of them—which are not characterized by the typical course described, and in which the initial stage is so prolonged that the latency is nearly or entirely omitted; or the same effect is brought about by the terminal processes beginning early and pursuing a very slow and insidious course. But we always see that the symptoms in the gross are the same, and that only their grouping, the rapidity of their succession, and the acuteness of their course, vary. As for the duration of these abscesses, they can without difficulty be reckoned among the chronic ones.

The cases with very protracted initial stage (from two to three months) are very rare, and their history during it is composed of a number of episodically occurring sets of symptoms which always make a new encephalitic advance probable: soporific confusion, headache, in some cases convulsive symptoms of local disease, in others absolute lack of the same; also a mixture of general symptoms of pressure on the brain with those of irritation at isolated points, or—for paralyses have been seen which increase and decrease in intensity—of total disturbance of function in a circumscribed portion of the brain. In some cases (Vollmer, Gull) a short latency showed itself after such protracted appearances, and was followed by a very short terminal stage similar to the episodic aggravations of the initial stage, which soon ended in death. The latent stage may, however, be entirely wanting, and then a constant succession of brain-symptoms appear which make it doubtful whether the case should be classed among the acute or the chronic abscesses of the brain.[1]

II. *Chronic abscess in affections of the inner ear.*—Concerning this variety of abscess of the brain, it must be said that only a small part of the cases follow a course which allows a relatively certain diagnosis. Two points especially are to be kept in mind: chronic abscess of the brain is found after a cerebral affection which has appeared suddenly and ended fatally, and which must be regarded clinically and anatomically as the terminal stage; only intimations of an initial inflammatory stage are found during life, and even these may be absent. It appears, therefore, that this variety of chronic abscess of the brain may begin without any serious symptoms whatsoever, and may grow for a long time without giving rise to any material general disturbance resembling acute encephalitis; such abscesses also appear to possess a capsule from the commencement; we have seen at least one abscess furnished with a resistant capsule and measuring half a centimetre in diameter, filled with thickened pus, on the end of the temporal lobe, in caries of the cavity of the tympanum, which had caused no symptoms at all. The second circumstance which renders the recognition of abscess of the brain essentially difficult in these cases, is the resemblance which its terminal stage may show to diffuse meningitis, or even to certain cases of thrombosis of a sinus. In point of fact, confounding terminal meningitis with abscess of the brain is not always to be considered an avoidable error of diagnosis; and, finally, both can occur together, as when the abscess extending to the surface excites a meningitis which leads quickly to death; in such a case the latter is the cause of death, and if the abscess had not lain so near the surface it might have remained latent and been supported still longer. We shall consequently have to describe separately a number of varieties, the recorded accounts of which it is exceedingly difficult to find perfect and in sufficient number:

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[1] *Casper's* Wochenschrift. 1844, 13.

a. Chronic abscess of the brain with distinct typical course.

b. Chronic abscess of the brain, with terminal stage alone distinct.

c. Abscess without typical course, with a continuous series of brain-symptoms analogous to atypical traumatic abscesses.

d. Old abscess, with fresh thrombosis of the sinus.

Acute abscess has already been disposed of.

45. A factory girl, seventeen years old, with a discharge from the right ear since she was twelve years old, which is said to date from an acute disease (typhoid fever). In the course of this ear-affection, but especially at the commencement, much earache; gradual failure of hearing in the right ear. Four months ago had (beginning of July, 1870) an acute disease, which was regarded by her physicians as meningitis. This lasted about ten days. It began, without the discharge from the ear having ceased, with severe headache in the entire head, dizziness, sleeplessness, and mild delirium; slight chill and fever are also said to have been present; likewise vomiting several times. In twenty-four hours the patient had become completely unconscious, snored, drew the head back into the pillow (contraction of the muscles of the back of the neck), passed her urine and fæces in bed, and was given up. Occasionally consciousness was partially restored, but never entirely during the first ten days. In these intervals, which lasted for several hours, more or less delirium was always present. Nothing is to be learned of symptoms of local disease; at all events, neither paralysis nor convulsions were present at that time, nor was there unilateral paralysis of the face. Contortions of the eyes and their convulsive displacement toward the left are said to have existed. Contrary to all expectation, the meningitis improved after the tenth day, the patient became clearer, gained consciousness again, but always complained of severe headache and dizziness. An intense headache in the occiput, and pain on turning and shaking the head, remained for a very long time.

Up to July 20th, when patient came under observation, she is said to have been well, except for frequent and severe attacks of headache, which lasted a few hours, were associated with reddening of the countenance and of the conjunctiva, and appeared every three or four days, especially in the back of the head. Nothing like a convulsion was ever seen; also no severe attacks of fever and the like. During the entire time she was very thin and weak, and for this reason incapable of work. The discharge from the ear had never ceased, and was even at times stronger than ever. The headache was especially severe during the days just preceding the 20th of July.

On the 20th of July, while the patient was working without specially exerting herself, she was suddenly attacked with an exceedingly severe headache and dizziness. This time she lost consciousness, and fell to the ground. No convulsions; no distinct paralyses; reddened countenance; slow, snoring respiration. She lay two hours unconscious, and then slowly recovered. Since then only complaints of very severe headache, dizziness, whizzing in the head; speech and movements undisturbed; no sleep.

21st.—Present condition: a lean, poorly nourished girl; no cicatrices from glands; no signs of scrofula or tuberculosis; discharge from right ear (perforation of membrana tympani, and apparently, though not accurately determined, loss of hearing in right ear). An hour ago the patient fell back into her unconscious condition: quiet decubitus, with a somewhat prolonged, gently snoring respiration, closed eyes, reddened face; can be roused out of her unconscious condition by energetic excitation, but not to entire consciousness; then she opens her eyes. Right pupil enlarged, reacting badly; the left also reacts feebly, and is moderately dilated; no ptosis; no cramps of the eye-muscles; no axial deviation of the eyes; nothing in the facialis or hypoglossus. All the extremities movable; no difference between the two sides; sensibility retained; reflex irritability of the legs increased. Severe general headache; hyperæsthesia over the entire body; vomiting. Temperature 38.8° (101.84° F.). Pulse 112, small.

22d.—Unconscious the whole night; this morning, deep sopor; occasional mild delirium; pupils equal; cramps of the muscles of the eyes; eyes turned up and toward the left; no paralysis of the facialis or hypoglossus; reflex acts of swallowing still made. Vomited twice; no stool. Slight tension of the muscles of the neck; bending of the spinal column painful. Nothing in the extremities. Temperature 39° (102.2° F.). Pulse 108.

At eleven o'clock in the morning, an epileptic attack. Twitchings begin in the left arm, go over to the left leg, the left facialis; eyes toward the left; then they appear in less degree in the right arm and leg, cease after a few minutes in the last locality, and in about five minutes afterward end on the left side. After the convulsion there is sopor, with snoring, and entirely reactionless pupils, but no paralysis.

Evening. Temperature 39° (102.2° F.). Pulse 88, full.

At ten o'clock in the evening the convulsion reappears, is less severe, and follows the same course.

23d.—The entire night, sopor. This morning it is seen that the left pupil has become equally dilated with the right; both fail to react. On the left side, ptosis and slight divergent strabismus; slight paralysis of the left facialis and hypoglossus. Movements are made with all the four extremities, but those of the left are feebler. No more convulsions; no vomiting; no increased stiffness of the neck-muscles; no retraction of abdomen. Retina shows considerable general congestion, and on both sides the veins project over the swollen papilla.

Temperature 38.4° (101.12° F.). Pulse 108, small.

Deep sopor, lasting until evening; no convulsions.

24th.—Similar condition; no change in the nervous symptoms; tracheal râles. Temperature 39° (102.2° F.). Pulse 116, small.

Death at four o'clock in the afternoon.

Autopsy.—Pia and convexity normal and anæmic; marked general swelling of the brain. Dura and pia, together with the brain (right temporal lobe), adherent over the roof of the cavity of the tympanum, by old adhesions, to a considerable extent; no suppuration there or in the neighborhood. Ventricles not distended. In the right temporal lobe an old encapsulated abscess; capsule, two and one-half to three millimetres thick; pus of usual character, not putrid. Around the capsule a broad zone, especially on the upper side, of red inflammatory softening, extending into the base of the nucleus lentiformis. The entire temporal lobe, the ganglia of the brain, and the right hemisphere up to the level of the corpus callosum (Balkenniveau), very œdematous. Many small apoplexies in the zone of red softening. Caries of the wall of the tympanic cavity; the upper wall not perforated. (Our own observation.)

This case can serve as a typical proof of the existence of chronic abscesses, in caries of the petrous portion of the temporal bone, which take the characteristic course of traumatic abscess; at the same time it is apparent that the cause of death may also be a fresh encephalitis (etiology unknown) around the abscess, which kills by producing great pressure upon the brain. The symptoms of local disease are of interest, inasmuch as the convulsions were stronger on the left side, and the paralyses appeared sub finem, which were evidently to be ascribed to the spread of the encephalitis to the base of the nucleus lentiformis. Since this is not the case in all abscesses, the occurrence of paralyses must, of course, not be expected in all cases; many other cases do not show them.

We still more rarely find abscesses of the brain in caries of the petrous bone, which are to be classed, anatomically, as chronic, and cause death by virtue of their effect on the brain alone, or by a complication, without any cerebral affection, which could be interpreted as primary encephalitis, having ever preceded. The absence of symptoms must here also depend on the locality of the abscess, for, if the abscess be not large, it is wholly impossible that hemiplegic disturbances or other permanent symptoms of local disease can be called out by it when occupying the usual position in the temporal lobe. The loss of brain-substance in the temporal lobe, which abscess of the brain necessarily produces, is not sufficient according to experience to call forth important symptoms.

Those abscesses of the brain which are found associated with thrombus of a sinus are very obscure in their relation to the latter. In some cases it is certain that the formation of the thrombus has preceded that of the abscess, and it is probable that it has caused the suppuration of the brain by its puriform breaking-down. If one remembers that in thrombosis of the transverse sinus, for example, the coagulation continues up to the small veins of the pia, and even into the brain, and further that the puriform destruction begins there where the most extensive disease of the wall took place, and that it may spread in certain veins back even to the finest branches, then there is nothing incomprehensible in the formation of abscess of the brain, although it is true that many intermediate links escape our knowledge; in these cases, however, the abscess is an acute one. The opposite may also occur, and to a chronic abscess of the brain may be added a thrombosis of the sinus, which, however, does not depend on the abscess, but on the spread of the inflammation from the petrous bone; more intimate connections between chronic abscess of the brain and thrombosis of the sinus, at least, are not known.

Very many cases of thrombosis of the sinus, with abscess of the brain, have been found, which did not betray themselves during life by distinct symptoms, while other cases of pure thrombosis gave rise to a series of symptoms not difficult to interpret. We have come to the conclusion that the recognition of co-existing abscess of the brain and thrombosis is possible only in a very small number of cases. The course of these cases differs essentially from the sketch briefly given above of a typical case of abscess of the brain. Although the symptoms certainly vary in intensity, the course of the disease is not characterized by any clearly marked remissions. The duration usually extends over several weeks, but can vary within pretty broad limits, owing to the fact that the commencement of the changes is not marked by any characteristic symptoms. Dizziness and headache are the chief initial symptoms, and can increase to an unendurable degree. The sensorium is usually affected, though not to a great degree; occasional somnolence—and now and then transitory delirium—is not rare. Symptoms of the local ear-affection coexist. Usually the patient becomes feverish, and sometimes real chills have been observed, which may occur at any time during the whole course. The symptoms may then take either one of two directions:

Either in the subsequent course the symptoms of abscess of the brain preponderate, pressure is made upon the brain—at first varying, afterward more constant, but always progressive, and combined with symptoms of local disease as soon as the abscess or accompanying encephalitis has reached motor regions. General convulsions usually betoken the near approach of death.

Or else the signs of thrombosis of a sinus are more apparent, together with headache, unilateral flushing and swelling of the lids, unilateral exophthalmus, unilateral œdema of the cheek, of the temporal region, the mastoid region, unequal distention of the small peripheral veins (frontal vein, veins on the processus mastoideus), unilateral injection of the conjunctiva; finally, the known venous symptoms in the neck, incomplete distention of the jugularis externa, thrombosis of the jugularis interna, evident to the touch.

Or, finally, the symptoms of both affections unite, as in the following case of Wendt:

46. A man, twenty-four years old, in June, 1866, had left-sided otorrhœa, difficulty of hearing, and left facial paralysis; from his eighteenth to his twentieth year suppuration of glands. Since then intense headache, sometimes on the right side, sometimes on the left, and at times in the forehead; for the last year and a half difficulty of hearing and continued buzzing in the left ear; for the last six weeks otorrhœa and left facial paralysis.

Power of hearing considerably diminished on both sides; left facial paralysis; active and passive movements of the lower jaw very painful; right auditory canal and membrana tympani normal; in the left, a few polypoid growths, and on its posterior wall discolored, rough, bare bone.

The left ear was treated continuously for two years, and a relative cure with considerable improvement of function was obtained.

August, 1866.—Severe pain in right ear, swelling of the passage, great diminution of hearing; severe pain at different points on the right side of the head; discharge of offensive ichor from the Eustachian tube and auditory canal; an accurate diagnosis of the condition of the ear not possible, because the canal was almost closed by the swelling. After this had lasted two years the discharge of pus from the mouth, nose, and ear was very abundant; at the end of the year 1866 hearing in the right ear was entirely lost, the movement of the lower jaw became again very painful.

March, 1869.—Admitted to hospital; ptosis on the right side, redness and swelling of the lid, right exophthalmus, swelling of the right cheek and temple.

May.—Paralysis of the right facialis, swelling of the entire right side of the head, swelling of both lids and ptosis of the right. Fluctuation at several points.

Beginning of June.—Great exhaustion, intense pain in head and right ear, troublesome buzzing of the ears, almost complete deafness, profuse discharge.

June 20th.—Twitchings of the neck and of the extremities, lasting for half an hour; unconsciousness, death.

Autopsy.—In the right parietal, temporal, and frontal regions an abscess. Anterior half of the right temporal bone, squamous portion, necrotic. Beneath, the dura mater of the right hemisphere in its front half grayish-green to blackish; in the right frontal bone a roundish loss of substance, one-half inch in diameter, leading to the cavity of the abscess. At the base of the brain the dura on the left side is grayish-yellow; on the clivus, on the sella turcica, on the right half of the anterior, and in the anterior three-quarters of the middle fossa on the right side, it is thickened, grayish-brown to grayish-green, lifted from the bone by ichor. On the right pyramid the dura is grayish-yellow; in the right sinus cavernosus an old parietal clot. Convolutions of the brain slightly flattened, anæmic; in the right frontal lobe a cavity the size of a hen's egg, with brittle walls and blackish-gray, offensive, ichorous contents. In the front wall a hole the size of a silver five-cent piece, corresponding to the above-mentioned lesion of the frontal bone, through which the abscess of the brain communicates with the external purulent collection. Body of the sphenoid bone, right lesser wing of the sphenoid, outer two-thirds of the greater wing, and also the right pterygoid process carious. Caries of the processus zygomaticus of the right frontal bone, likewise of the orbital portion. Right condyle of the lower jaw carious. Very extensive caries of the petrous portion of the right temporal bone.

The following case of Wreden is very remarkable, not only on account of the perforation of the abscess of the brain externally, but also on account of the symptoms manifested during life.

47. A soldier, twenty-one years old, hard of hearing, and afflicted with slight discharge from both ears. Eight to nine weeks ago admitted to hospital on account of severe headache. Membrana tympani on both sides destroyed; since childhood otitis media; great pain in the left processus mastoideus. Death occurred in four weeks (entire duration from twelve to thirteen weeks).

The patient lay all the time in the left lateral position, crying on account of the severe headache. The pulse had become excessively slow—fifteen and even ten in the minute. Left pupil constantly contracted; no facial paralysis. A week before death increase of the discharge from left ear, and sudden improvement (discharge of the abscess of the brain ?), but soon followed by soporific condition and death.

Hyperæmia and œdema of the brain; exudation in the ventricles. At the base the meninges are adherent to the left petrous bone, and on separation pus flows out of the brain. The convolutions of the left hemisphere compressed. In the left hemisphere an abscess the size of a goose egg, which had emptied itself through the squamous portion into the region of the root of the zygomatic process, under the temporal muscles, as far as the articulation of the jaw. On the left side, caries of the roof of the tympanic cavity, a broken-down, purulent thrombus of the transverse sinus extending to the bulb of the jugular. Mastoid cells filled with caseous pus.

Both cases show the very exceptional occurrence of perforation of the abscess externally. The second gives the only instance, in abscess of the brain, of retardation of the pulse to fifteen and ten in the minute.

Abscess and meningitis in caries of the petrous portion of the temporal bone.—If all chronic abscesses ran their course according to the above-described typical picture, it would not be necessary to waste words on the differentiation of chronic abscess from meningitis. We have proved, however, that an initial stage is often enough wanting, and so the terminal stage may alone come under observation and completely imitate a rapid meningitis. Single cases of meningitis—see, for example, Cases 12 and 13 of Wendt (Arch. d. Heilkunde, 1870)—are characterized by nothing but quickly occurring loss of consciousness, snoring respiration, a few general convulsions, and early death. Signs of a quickly appearing and progressively increasing internal pressure, which leads at first to rapid compression of the cerebrum, then of the spinal cord, and finally of the medulla oblongata. And we find exactly the same course and symptoms again in abscess of the brain.

The differentiation of acute abscess of the brain and meningitis is just as uncertain. One must keep in mind that a larger number of cases of meningitis are characterized by a more protracted course, and that, like an acute encephalitis, they show sundry variations in intensity (see p. 613). Further, these cases run their course with convulsions, with paralyses, with stiffness of the neck (p. 615). Headache, fever, vomiting, cannot be taken as characteristic general symptoms of meningitis. Since now abscesses in the cerebrum, in caries of the petrous portion of the temporal bone, are for the most part, if not almost exclusively, abscesses of the temporal lobes, distinct symptoms of local disease are wanting in a very great number of cases. One therefore easily sees how similar the two pictures of disease must appear; for in an acute abscess of the brain thus situated, there will also be signs of a quickly increasing pressure—first within the skull, and then in the cavity of the spinal cord—to which pressure the different portions of the central nervous system oppose a different resistance: first, the cerebrum, then the spinal cord, and then the medulla oblongata succumbs. Distinct symptoms of localized disease (spreading of the abscess into the territory of the nucleus lentiformis), paralyses, hemiplegia, convulsions confined to single limbs and always repeating themselves there, are the best points to aid us in making the diagnosis.

Location of the abscess in the brain.—In this respect an extraordinary want of clearness still exists, and many observations appear entirely incomprehensible. It is in the consideration of abscess of the brain, therefore, that many authors have found opportunity to shake the apparently firm physiological laws.

Great collections of pus have been found in spots of which it is known that every other lesion causes a paralysis of the opposite side (medullary portion of the hemispheres); there exists indeed a whole series of cases of total suppuration of a hemisphere, of a frontal lobe, of the whole posterior lobe. It seems to us that all these observations have roused a wonder by no means justifiable; that the fact must be impressive we admit, but in none of the cases known to us have its causes been properly traced. For in no single case is it accurately proved what remained of the hemisphere—whether the inner capsule still existed, whether the continuation upward to the cortex was still preserved, or if it was consumed in the abscess. Our own observations as to the degree of consumption of the brain-substance lying around a chronic abscess, show that it is highly overestimated; the abscess does not consume the entire portion of brain whose place it occupies, but when it is encapsulated its action is rather a displacement with slowly following atrophy of the displaced portion. The only abscess of the frontal lobe which we had an opportunity to observe ourselves, belonged also to the number of those which appear to be stricken out of the usual scale; for it occupied almost exactly the place (under the front central convolution, directed downward toward the fossa of Sylvius) where, according to universal agreement, a focus must lead to paralyses. It had, however, caused none, and also no aphasia, although it occupied the right spot. But of this abscess at least we can surely assert that it had left intact the entire mass of fibres of the capsula interna and its upward continuation, which runs up between the nucleus lentiformis on the one side, and the thalamus and corpus striatum on the other side, and likewise the ganglia themselves. One always deceives himself about the power of this set of fibres of the brain, the most important ones in producing motion. An abscess in the frontal lobe can reach a really enormous size without injuring other systems of fibres as associate systems; by this and by the attenuation and atrophy of many portions of the cortex, room is obtained, so that even an extensive displacement of the system of fibres mentioned is possible without interruption of its functions. The greater the number of associate systems that have undergone destruction in an abscess of the brain, so much the more must the psychical processes suffer. This also is a point which in abscess of the brain has not by any means been sufficiently investigated; a number of patients show sufficient intelligence in the hospital to make them appear as if normal, but on account of weakness of their intelligence they have become incapable of pursuing their former occupation.

Similar reflections have been made several times before, concerning abscesses of the temporal lobe; when an abscess—and this applies also to the frontal lobe—by its increase really reaches and destroys motor territory, then motor symptoms certainly appear.

It is known that in recent times sensory functions are more especially ascribed to the occipital portion of the brain; a confirmation of this view is not to be obtained from the pathology of abscess. Pareses of sensibility and neuralgias are comparatively very infrequent in abscess of the brain ; the reason of this may lie in the extraordinary rarity of abscess of the brain in the territory of the ganglia of the hemispheres, and in the crus cerebri, lower down, where the motor and sensitive fibres run near each other. Meyer found distinct pareses of sensibility in only seven out of eighty-two cases ; only four cases of trigeminus neuralgia, of which two were on the same side as the focus. Certainly no one of all these was dependent on the direct action of the focus on the surrounding brain-substance. This author mentions sensation of pain in the paralyzed extremities only twice—anæsthesia dolorosa ; it is very questionable if it is to be regarded as a central symptom. When occipital abscesses grow toward the front and injure the inner capsule, which runs upward and inward from the nucleus lentiformis, hemiplegic symptoms appear.

We have hitherto not had the fortune to observe an abscess of the brain which had led to a manifest disturbance of speech ; most undoubted cases of aphasia exist, however, which have been caused by abscesses of the neighborhood of the left island (see the case of Boinet already cited, Gazette des hôpitaux, 1871 ; Sayre, Hammond's Diseases of the Nervous System, 1872 ; Lossen, Berliner klin. Wochenschrift, 1870). More cases refer to recent contusions of the brain (Nothnagel, Leyden [Berliner klin. Wochenschrift, 1867], Lohmeyer, Bergmann, Wernher, Benoit).

Besides aphasia, however, other different disturbances of speech have been observed. Meier, in his statistics, which reach to the year 1867, has twelve cases of disturbance of speech out of ninety ; one abscess was seated in the cerebellum, three in the frontal lobe, three in the occipital lobe, five in the middle lobe. Meyer regards this, as well as the known case of E. Schmidt, of suppuration of the whole frontal and temporal lobes, as a demonstration of the non-existence of an isolated centre of speech. When one considers that in the old observations accurate accounts of the kind of disturbance of speech are wanting in the great majority of the cases, that the disturbance of speech due to lesion of the medulla oblongata is entirely different from aphasia, that in very many observations all accounts of the peripheral muscles and their innervation are wanting; and when, finally, one fails to find in the old accounts of autopsies of abscesses of the cerebrum any accurate information concerning the relations of the abscess to the different parts of the hemisphere, he cannot well allow himself to base definite conclusions upon the existing data. The question of the localization of the power of speech is, moreover, one which most especially requires reserve in judgment, for the physiological value of what we know concerning the localization of aphasic disturbance has been considerably over-estimated, especially when one stubbornly insists, as some do, that it is connected with Broca's convolution.

Abscesses of the frontal lobe form about one-fifth of all the cases which have come to our knowledge. They lie partly within the motor tracts of the caudex cerebri (motorischen Stammfaserung), partly without it. About half the abscesses situated here cause distinct symptoms of localized disease; the other half are without them; the general course is the same in all.

The symptoms of localized disease are:

1. Hemiplegia, or hemiparesis, or paralysis merely of one extremity.
2. Isolated paralysis of the facialis, or coupled with unilateral paralysis of the body.
3. Paralysis of the hypoglossus, coupled with hemiplegic disturbances.
4. Contractures of the opposite side (very rare).
5. Isolated convulsions in the region of distribution of the facialis and hypoglossus nerves (gyrus, between the sulcus præcentralis and the fossa Rolandi).
6. Convulsions of the opposite side, involving the facialis, hypoglossus, and spinal accessory.
7. Diminution of sensibility on the opposite side.
8. Aphasia when the abscess lies within or near the walls of the left fossa of Sylvius and the connections running under the first frontal convolution to the motor region of the frontal lobe.

The general symptoms are:

1. Headache, in the large majority of cases; a small proportion of abscesses show a localized headache, which then corresponds to about the position of the abscess.

2. General convulsions; in the terminal stage in about one-third of the cases.

3. The disturbances caused by pressure on the brain in the terminal stage, concerning which, all that was necessary has been already said.

Abscesses of the temporal lobes.—These form a second fifth of all cases; they usually lie outside the motor tracts associated with the cerebral ganglia (Stammfaserung), but reach them by increasing upward, or by complicating encephalitis; then usually not only the base of the nucleus lentiformis, but also the adjoining capsula interna, is affected. As a result of this the symptoms of localized disease are more rare—about two-fifths of the cases; of the convulsions, those are wanting which are confined to fixed groups of muscles. The symptoms of localized disease are therefore:

1. Hemiplegia or hemiparesis.

2. Facialis- and hypoglossus-paresis likewise, but never isolated.

3. Contractures of the opposite side (rare).

4. Paresis of sensibility of the opposite side.

Among the general symptoms headache is the chief; it is present in all cases with few exceptions, and is of considerable intensity. Sometimes it was confined to the front, sometimes to the back of the head. The remaining general symptoms are the usual ones.

Abscesses of the occipital lobe form about one-eighth of all abscesses of the brain. Symptomatically, they are of all abscesses well-nigh the least clear. The almost entire absence of all disturbances of sensibility is striking. The symptoms of localized disease (one-third of the cases) are:

1. Hemiplegia and hemiparesis. The mode of production of these symptoms has never been anatomically explained; a close examination has never been made for the purpose of showing how far forward the focus had advanced into the ganglia of the brain, or to what extent the latter were directly or indirectly injured.

2. No isolated convulsions of the opposite side, but general convulsions commenced with twitchings in the extremities opposite the focus.

3. Contracture of the opposite extremities (very rare).

Among general symptoms headache is very constant; general convulsions occur in somewhat more than a third of the cases. All other general symptoms correspond to what has been already said.

Abscesses of the pons have been seldom reported (Wendt, Forget, Meynert). Wendt's case was a child with multiple periostitis also of the skull. Symptoms of local disease were: ptosis on the left side, and contracture on the right in elbow- and finger-joints, also cross-paralysis (oculo-motorius—extremities). There was an abscess in the corpora quadrigemina. We place this case, on the authority of Wendt, among the abscesses, though the description rather gives the impression that it was a caseous nodule.

In Meynert's case there was right-sided facialis- and oculomotor paralysis (yet only of the branch to the levator palpebræ), and contracture of the masseter. In the transverse inner layer of fibres of the pons was an abscess with devious extensions; it had broken both into the fourth ventricle and at the base. The perforation into the fovea rhomboidea (floor of fourth ventricle) had brought about facialis paralysis and irritation of the portio minor of the trigeminus. The longitudinal fibres of the pons were spared, or at most somewhat pushed over to one side.

Abscesses of the *cerebellum* form an entirely separate class.

Of the symptoms the following deserve mention:

a. Headache; this in abscess of the cerebellum becomes exceedingly severe, and is usually a prominent symptom during the entire duration of the disease. Its seat is usually the occiput, rarely the frontal region; occasionally the whole region of the neck and cervical vertebræ is highly painful. This corresponds perfectly with the cephalalgia accompanying tumors of the cerebellum.

b. Vomiting; in abscess located at no other point is this so distressing and obstinate. It is, indeed, not absolutely constant, but yet is present in the large majority of cases.

c. Disturbances of the gait. In this respect abscesses of the cerebellum have a great resemblance to tumors of the cerebellum. By some the disturbance of the gait is designated as ataxic

(Feinberg, Wilson), and it is not to be denied that it closely accords with it in form; we have had an opportunity to convince ourselves that the disturbance of the gait is seen without a determinable diminution of strength in the legs. The legs could be moved in bed with ease without showing any lack of co-ordination.

d. Of all abscesses of the brain those of the cerebellum most seldom show symptoms of localized disease. We know of only two cases where hemiplegia is reported, and even in these it is not proved that the lesion was not one of the medulla oblongata.

Isolated convulsions appear never to have been seen; so far as convulsions occurred, they were general, and in a small minority of cases only. Partial convulsions of the muscles of the extremities are also wanting; if contractions were present, they were those of the muscles of the neck or of the back.

Characteristic disturbances of sensibility are just as little to be found in abscesses of the cerebellum as disturbances of motion.

e. Disturbances of the mental functions. Abscess, like tumor of the cerebellum, is characterized by greater rarity of psychical disturbances; even during its acute episodes, during convulsions, the sensorium is found completely free (Andral). This might nevertheless belong to the exceptions; for in the terminal stage the sensorium suffers in the majority of cases, just as in other abscesses of the brain.

f. Mydriasis, even bilateral, has been observed several times in abscess of the cerebellum. The rest of the muscular apparatus of the eye remains completely normal, so that paralysis of both oculo-motor nerves cannot be suspected. Probably it depends on an irritation of the sympathetic fibres of the pupil starting from the medulla oblongata.

g. A few cases show paralysis of the abducens.—It causes a strabismus convergens, and in this respect also abscess coincides with tumor.

h. Disturbance of speech is a very rare symptom. Unfortunately, we do not know the kind, but it can scarcely have been aphasic, and was probably a disturbance of articulation.

i. Abscesses of the cerebellum resemble tumor still more closely in their *more continuous course.* A distinct latent stage can be made out much more rarely than in abscesses of other localities. The chronicity is not affected thereby; but the symptoms are either present constantly, with variations in intensity, or they form a series whose intervals in length differ very little from each other. The reasons for this variation are unknown.

Since one would hardly determine to diagnosticate an abscess of the brain without being certain of the etiology, it is well to remember the valuable diagnostic points which the latter gives in relation to suppuration of the brain.

If an injury has been received upon the head, the abscess, in the majority of cases, lies near the seat of the injury; the cases are rare where an abscess is formed on the other side of the brain by contre-coup.

In cases of otorrhœa we know that the probability of an abscess of the cerebellum is greater when caries of the processus mastoideus is present (it comes occasionally from caries of the walls of the cavity of the tympanum and of the labyrinth). If there is merely caries of the tympanic cavity, abscess of the temporal lobe is the most probable; the latter almost always occurs from perforation of the roof of the tympanic cavity.

If there is caries of the bones about the nasal cavity, and an abscess of the brain is suspected, its situation must be sought in the frontal lobe.

In purulent ichorous affections of the lungs, in pyæmia, we can only say that these abscesses choose the medullary substance of the hemispheres, but otherwise have no place of predilection, so that in regard to this class of abscesses no conclusion is to be arrived at.

Conditions of the retina.—Disturbances of vision in abscess of the brain have been observed in a relatively small number of cases. Meyer, in his work, cites only five such cases, three of which (Batemann, Peacock, Gull) ended in complete double amaurosis; accurate ophthalmoscopic data are wanting. Add to this Webber's imperfect case of abscess around a glioma of the cerebellum. In two other cases cited by Meyer there was complete amaurosis on one side, dependent on periostitis at the entrance of the optic nerve into the foramen opticum (see Meyer's Case VI.).

As for simple congestion of the retina, compare the cases reported earlier, and also one of Meyer's, where, on the left side, the appearances of congestion in a case of abscess on the left side of the brain were considerably more marked than on the right, and apoplexies of the retina were present (examined by Horner). Ampulla on the optic nerve is mentioned in one of the above reported cases.

Neuro-retinitis duplex has recently been observed several times. The cases may be divided into two categories: in the one meningitis was present together with the abscess of the brain; in the other, not. Of the cases where meningitis was wanting, two of Jackson's are especially worthy of note (London Hospital Reports, IV.). Further accounts come from Peipers (meningitis present), Benedict, Maunder.

Finally, we know of a case of hemiopia which was not accessible to us in the original (Lewick, Americ. Journ. of Med. Sci., 1866); abscess in the left frontal lobe, and one in the right posterior lobe.

a. Symptoms of passive congestion in the retina.—It is easy to understand why we possess no accurate observations concerning the existence of this in the latent stage of abscess of the brain. The accurate investigations belong either to an initial stage, characterized by great cerebral disturbances, or to the terminal period; but that these episodes of the course are not the only ones associated with the disturbances under discussion is sufficiently evident from the neuro-retinitis to be spoken of later, which requires a long time for its development.

After what has been said, passive congestion of the retina in abscess of the brain cannot cause wonder. Any pressure on the brain may produce it; and, with reference to this, we have called attention especially to the pressing out of the fluid from the subarachnoid space into the sheath of the optic nerve. In the course of the abscess, opportunities enough are presented for it, and it is possible for transitory conditions of coma to cause transitory congestion. The fact is of great interest, that an inference as to the side upon which the abscess is situated may be drawn from the greater congestion of one eye as compared with the other. The ampulla on the optic nerve is the simple anatomical expression of increased pressure in the optic sheath.

b. Apoplexies.—Their occurrence in abscess of the brain has hitherto been very rare. They are attributable to obstruction of the venous circulation.

c. We do not possess accurate determinations of the central and excentric sharpness of vision in cases of retinal congestion. We must call attention to the fact, however, that in the large majority of cases we have to deal with patients who are out of their senses.

d. Neuro-retinitis.—The fact is important that this occurs both with and without meningitis. In the latter case, a propagation of the inflammation from the pia, through the subpial space up to the lamina cribrosa, cannot be maintained. Without wishing to deny this propagation in meningitic cases, we hold to the opinion that a simple venous congestion, caused by the abovementioned factors, is sufficient to give rise to the symptoms of neuro-retinitis. The nerve is compressed at the lamina cribrosa. The veins are first strangulated ; then the arteries ; in the meantime the papilla is elevated somewhat by œdema. Extravasation of the white blood-corpuscles follows swelling of the connective-tissue fibres in the neighborhood of the papilla. This is able to cause the characteristic appearances of neuro-retinitis, and even partially to cover up the signs of congestion (Schoen, Leber, Hulke).

As for the production of neuro-retinitis in those cases in which meningeal disturbances coexist, various opinions are held.

Diagnostic points are only rarely to be obtained from neuro-retinitis, for even without this the diagnosis is usually narrowed down to the supposition of tumor or abscess ; the riper experience of recent times, in fact, allows in those cases which are not meningitis only one conclusion, *that of increased intra-cerebral pressure of such a kind that marked congestion, with its consequences, must follow.*

If the affair is not soon terminated by the death of the patient, entire loss of sight and atrophia optici follow.

e. Hemiopia.—We know nothing of temporal and nasal hemiopia in abscess of the brain. The above-cited case of Lewick shows equilateral hemiopia, without our being able to demonstrate its origin from the anatomical appearances.

The occurrence of hemiopia from a unilateral focus of the brain is, however, comprehensible. The following case, although not directly belonging here, serves to demonstrate this point, and this is a suitable opportunity for its publication:

48. Woman, forty-six years old; had acute rheumatism fifteen years ago; since then, palpitation of the heart, and, for the last few years, loss of strength, cyanosis, frequent feeling of oppression, occasional hæmoptysis (infarct), chronic cough, and oftentimes transitory œdema; extreme stenosis of the mitral orifice.

About three months ago she suddenly fell down unconscious. A few slight convulsive movements were made on the right side of the face and in the right arm. The loss of consciousness lasted an hour and a half. After recovery she presented the following symptoms:

1. *Right-sided paresis of the extremities;* both limbs still movable to a slight degree.

2. *Paresis of sensibility of the right side* (arm, leg, body, right half of the face). Sensibility to pain very much diminished; localization bad; the capacity for perception of differences of temperature almost entirely destroyed.

3. *Paralysis of right hypoglossus and facialis,* latter confined to mouth and nasal branches.

4. *Marked aphasia.* A number of musical sounds are lost, the patient also cannot read and cannot repeat what is said. Writing impossible on account of the paresis of the right side.

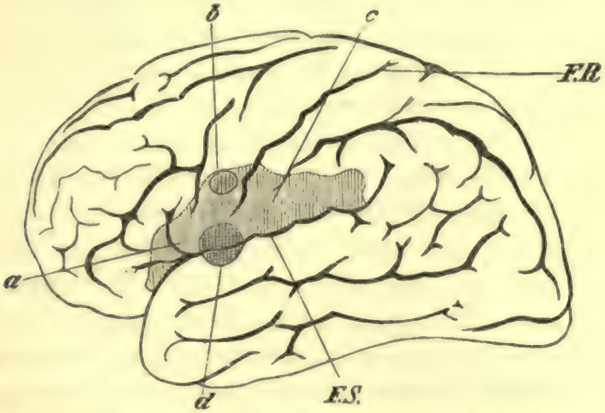
5. Confused and unsettled state, which, in spite of the aphasia, can be easily determined from the manner of the patient.

The dazed condition lasted about eight or ten days; her mind then became more and more clear, and she commenced in her awkward way to complain of affected sight, and after a time the field of vision was tested and showed *hemiopia of the right side. The left halves of both retinæ were deprived of the power of sight.* The line of separation is not accurately fixed; it does not appear to be entirely vertical.

In the other symptoms no sign of improvement ever showed itself. The heart grew weaker and the dropsy increased; death by paralysis of the heart followed three months after the embolus.

Autopsy.—Artery of the left fossa of Sylvius closed by a firm plug from the point of origin to the place where it bends over on the cortex of the island. Necrosis of the following portions of brain:

1. Broca's convolution (first frontal convolution of the modern, third of the old notation), *a* on the accompanying figure.
2. Hitzig's centre for facialis and hypoglossus (gyrus præcentralis), *b*.
3. The point *c behind the fossa Rolandi*, up to near the end of the posterior ascending branch of the fossa of Sylvius, *c*.
4. The upper parts of the fan-shaped small gyri of the cortex of the island.



a. First frontal convolution. *b*. Hitzig's centre. *c*. The spot claimed for hemiopia. *d*. The destruction described by Wernher (Virchow's Archiv. Band 56). *F.S.* Fossa of Sylvius. *F.R.* Fossa Rolandi.

The necrosis goes deepest into the brain in the region of the convolutions of the cortex of the island. Outer wall and outer member of the nucleus lentiformis are partially destroyed; on the spot *c* the necrosis is two centimetres deep.

Stenosis of the mitral valve; infarction of the lung; cyanotic induration of the liver and kidneys; general œdema.

Several such cases have been already described, and Schoen has determined their physiological significance (Schoen, Derby, Berthold, Keen, and Thomson). It appears from the short sketch given above that *behind the sulcus Rolandi lies a spot in the cortex, injury of which causes hemiopia. Still farther behind, a centre for the sensibility of the surface of the body appears to lie.* Whether the corresponding centres lie only in these spots is at present entirely unknown. In view of the radiation of the connections of the posterior tuberosity of the thalamus opticus and the corpora quadrigemina, also of the corpora geniculata, it must be presumed that the expansion of these centres is very great.

This conception of the affair presupposes the semi-decussation of the optici; if the believers in total decussation should win the victory—which, however, is scarely probable—then a re-crossing in the region of the corpora quadrigemina must be supposed. We have no anatomical knowledge upon this point.

Diagnosis from other diseases of the brain.

1. The symptoms of abscess of the brain have in course and grouping most resemblance to those of tumor of the brain. This must be obvious when one recalls the nearly identical demeanors of the two foci; both exert a certain pressure upon the surrounding brain-tissue and cause it to disappear; both are occasionally at rest for a long time, which, however, is more marked in abscess than in tumor of the brain; both cause consecutive changes of the adjoining parts—inflammatory disturbances, which are often more important, as regards both symptoms and life, than the focus itself. Both are subject to the same law with regard to location, namely, that if they occupy certain places they do not necessarily cause any symptoms of localized disease, but may present only general symptoms during the entire course. Finally, in rare cases, abscess may be superadded to tumor, as has been already explained, so that the symptoms unite in a common picture of disease.

I. The etiology of the two diseases is different.

It is known that injury also plays a part in tumor of the brain, as was demonstrated by Griesinger; although this is always rare, it should be borne in mind when making a diagnosis. Attention has already been sufficiently called to the fact that injury forms the chief etiology of abscess of the brain. As for the quality of the injuries, all that is necessary has been said above. Abscess under such circumstances is the easiest and surest to diagnosticate:

a. If an acute disturbance of the brain follows an injury, which accords with the symptoms of acute encephalitis (see above).

b. If an abatement of the symptoms amounting nearly to restoration of the normal condition takes place: latency. Imperfect latency has been described above, with its different symptoms.

c. If finally, after the latency, a new acute cerebral affection of the brain breaks out, which bears the character of a rapidly increasing focus, exerting great pressure on the adjoining parts, and if this affection leads to death. The attack, however, may pass over, a second latency occur, and the third then terminate life.

A tumor developing after an injury to the skull would never follow such a course.

Chronic otorrhœa also plays a part in tumor (Fischer). It is, however, quite as subordinate or even less important than that of injury. It has been shown several times before that otorrhœa can have its origin in very different affections of the ear. Carious destruction of the neighboring bones—the squamous portion of the temporal bone, the wing and body of the sphenoid—occurs in addition to disease of the petrous portion of the temporal. As soon as a disease of the brain, which presents distinct symptoms of localized disease, but never the appearances of congestion characteristic of thrombus of a sinus, is added to one of the above-mentioned affections, the diagnosis of abscess of the brain can be made.

In injury and otorrhœa it must be remembered that the course can be acute and chronic. In otorrhœa the acute course of the abscess is the most common occurrence. It leads, in three or four weeks, to death; and under these circumstances cases with distinct appearances of local disease are not difficult of diagnosis. We have spoken above of its differentiation from meningitis; the differentiation of marked cases of thrombosis of a sinus is also not difficult.

One circumstance, however, in this connection has led to error in the diagnosis. There are abscesses of the brain of acute course with no symptoms of localized disease (temporal lobe, cerebellum); among these, there are some with chills and high fever. On the other hand, there are thrombi of a sinus without characteristic vascular symptoms (simple congestion in the retina occurs in both, and, therefore, is not conclusive), but with pyæmic chills and diffuse brain symptoms. There are some cases where a diagnosis is simply impossible. Finally, the different combinations furnish new difficulties of diagnosis (abscess with meningitis, abscess with thrombosis of sinus, meningitis with thrombosis).

If the abscess is recognized, its symptoms may diverge in two directions, namely:

a. No appearances of localized disease appear, but a general ("ataxic") disturbance of the gait; a very intense, constant, occipital headache; intractable vomiting; finally, general symptoms of pressure, epileptiform convulsions, and stiffness of the neck. This is abscess of the cerebellum.

b. Distinct symptoms of local disease, hemiplegia, partial convulsions of the opposite side, general severe symptoms of pressure, convulsions of a general nature. This is abscess of the temporal lobe, which has progressed up into the motor tracts connected with the cerebral ganglia (Stammfaserung).

Chronic abscess in otorrhœa in its typical course has been previously described; there are cases whose course does not differ from that of chronic traumatic abscess.

The middle forms, standing between the two, have been mentioned above; they are to be judged according to the principles previously laid down.

The carious processes in the front part of the cavity of the skull, and of the bones around the organ of the sense of smell, have no importance in relation to tumor; they can, however, lead to abscess of the brain, and the symptoms then caused are to be judged of as in analogous affections due to caries of the petrous portion of the temporal bone. The exceptional relations of the frontal brain must be well kept in view; but it must be remembered at the same time that direct disturbances of motility are produced by injury of only a part of the fibres of the frontal portion of the brain.

Chronic pulmonary affections, especially bronchiectasia and purulent cavities, in which putrid degeneration of the secretion occurs, have no relation to tumor. We have referred above to the difficulties of distinguishing abscess of the brain, especially from tuberculous meningitis. They depend upon the fact that not all abscesses of the brain cause distinct symptoms of local disease.

2. The course of chronic abscess of the brain does not correspond to that of tumor. The course of the latter is characterized, to be sure, by remissions, but they are frequent, while in abscess only one, or at most two, come under observation. After every remission of the symptoms, the course of the disease, in tumor, becomes more severe, for new symptoms appear ; to the headache are added disturbances of motility ; to these again, those of sensibility and of special sense. Moreover, there are cases where remissions do not occur at all, but the entire course is continuous and progressive. It is easily seen from the previous accounts of the extreme variations in the duration of abscess of the brain that the duration of the disease cannot be considered as a decisive point ; finally, it has been pointed out that there are chronic abscesses which arise without symptoms, where initial stage and latency occur together, and only the terminal stage comes under observation.

3. The special symptoms are, in truth, most useless for the differentiation of the two diseases ; in fact, they are so harmonious that not their quality, but merely their succession and grouping, may aid in forming a conclusion.

Psychical disturbances, especially increasing mental weakness, indicate tumor rather than abscess ; in the latter the psychical condition during the acute period is, of course, abnormal ; in the latent stage, also, it is rare that some psychical trouble, though it may be slight, is not present ; but steady progress in the disorganization from week to week, without nervous disturbances pointing to a complicating incident, occurs more frequently in tumor than in abscess.

In the differential diagnosis of the two diseases cephalalgia does not count for much ; it is said that it possesses a more paroxysmal character, that relatively free intervals occur, and that it is more severe in tumor than in abscess. The latter certainly, in its acute episodes, shows a continuous cephalalgia, but nearly complete remissions, similar to those seen with tumor, occur in the latent stage ; consequently these distinctions, which are not always valid, must be used with great prudence.

Conditions of high fever and chills are more indicative of abscess than of tumor. There has seldom been any reason to confound the latter with intermittent fever. The encephalitic complication so common with tumor, and which may cause intense fever, must be borne in mind; and we have also seen a chill, the cause of which was never made out, in a case of tumor.

The rarity of disturbances of sensibility in abscess is striking; in tumor they are more frequent, both as an isolated sign of localized disease, and in connection with motor paralyses. It is evident that a decision in any individual case could not be based upon such a point.

Motor paralyses are rarer in abscess of the brain than in tumor. It is self-evident that similar laws govern for the existence of paralyses in one as in the other; if, however, tumors and abscesses occupying nearly the same position are compared, paralyses are found to be more frequent in tumor.

Isolated convulsions are not available for differential diagnosis; they occur as well in abscess as in tumor, and with about equal frequency.

Just as little do epileptiform convulsions furnish a sure diagnostic point. Their frequency, severity, and special form cannot be used for diagnosis. When hemiplegia remains after an epileptiform attack, it is said to indicate abscess rather than tumor; our own observations do not confirm this statement.

The disturbances of the organs of special sense, especially the sense of sight, are more frequent in tumor than in abscess. Yet, in the latter, observations in point have increased in number in the last few years. A more accurate settlement of this point will perhaps essentially modify our conclusions in the future.

The most valuable distinctive points are, at all events, to be drawn from the etiological relations and from the course. It is easy to see, however, that there are cases, and even a superficial consideration of the recorded cases confirms this, where a sure differential diagnosis is impossible.

The terminal stage of abscess in itself, without an accurate knowledge of previous occurrences, may, under certain circumstances, be impossible of diagnosis. If an acute encephalitis occurs around an abscess, rapidly consumes the neighboring portions of the brain, and leads, when the abscess occupies a favorable position, to hemiplegia, contractures, and convulsions of a partial nature, ending quickly in the development of extensive œdema of the brain, the condition is favorable for diagnosis in this respect, that a preceding lesion of the brain which, according to experience, can lead to an acute encephalitis, must be supposed to exist (focus of softening, tumor, abscess); for we do not with certainty know of such a thing as primary acute encephalitis. Here the history of the case will aid greatly in the differentiation. If this cannot be had the diagnosis will remain obscure. As soon, however, as abscess with only general œdema of the brain leads to death, important symptoms of localized disease being absent, the diagnosis can perhaps be occasionally made, with the aid of an accurate knowledge of all that has preceded (injury, initial encephalitis, latency !); in the opposite case, however, as the experience of the best clinical observers proves, it will scarcely go beyond the recognition of an acutely occurring great pressure on the brain. Under certain circumstances the picture may be a complete meningitic one. One who is experienced in the polymorphous appearances of acute meningitis will, in such a situation, certainly think of abscess of the brain, but will guard himself from overstepping the bounds which now limit our knowledge.

If the abscess breaks through into the ventricle, the terminal stage can comport itself as in a large acute apoplexy. We have even seen several embolic infarctions with considerable hemorrhage, which broke through from the region of the ganglia into the ventricle—that is, which burst the thin wall between the focus and ventricle, and caused the symptoms of perforation of an abscess—exactly as above described—and were also diagnosticated during life as ruptured foci. If, however, the antecedents of the patient are not accurately known, the differentiation here is impossible.

If the abscess pursues a typical course, and if it can be accurately followed from the beginning, it is of course impossible to mistake it for apoplexy and embolism.

Thrombotic softening (also embolic) with encephalitis of the surrounding parts may, under certain circumstances, be very difficult to distinguish from abscess of the brain. Here we must again call special attention to the peculiar course of very many abscesses of the brain—a course which is not observed in the usual encephalomalacia. Secondly, the etiology must be considered: encephalomalacia depends almost exclusively on chronic diseases of the vessels, which play only a very subordinate part in abscess; embolic necroses also are sometimes connected with disease of the vessels, but far oftener with disease of the heart. Embolic abscesses of the brain are exceedingly rare. Injury, otorrhœa, putrid pulmonary affections have only very subordinate relations to real necrosis of the brain. The periods of life at which the diseases are observed have a bearing upon this also. In the years between fifty and seventy, where softening is so frequent, abscess is very rare; embolic softening occurs usually in early and middle life.

Furthermore, in certain cases the symptoms may be very similar:

Hemiplegic disturbances are more frequent in softening than in abscess, on account of the peculiar arrangement of the arteries which supply the region of the motor ganglia. If one compares an equally large number of softenings and abscesses, many more of the former than of the latter are found which involve the motor cerebral ganglia, or tracts connected with them (Stammfaserung) from before inwards.

If a sufficient reason for the preponderance of paralyses in softening can be found, it is not the case also with contractures; they are also considerably more frequent in softening—a fact which has not yet been satisfactorily explained. It appears to us that the nearer the softening lies to the cerebral ganglia, and the more the latter are themselves affected by the advancing softening, so much the more frequent are the contractures; which would correspond to the more frequent situation of softening in the deeper parts.

The fact is just the opposite in isolated convulsions. They are much more frequent in abscesses of the brain than in softening. We are under the impression that the convulsions confined to single groups of muscles are the more frequent the nearer the abscess approaches the cortex and the more it involves it. This corresponds also with the analogous situation of the tumor. General convulsions are much more frequent in abscess than in softening. Disturbances of sensation are much more frequent in softening than in abscess.

Headache is more prominent in abscess than in softening; in the latter it is often slight, and confined to occasions when inflammatory complications exist in the neighboring parts.

Finally, softening is accompanied, much more frequently than abscess, by psychical disturbances, weakness of memory, weakness of judgment, inability to think and decide, also by conditions of excitement, restlessness, sleeplessness, hallucinations and temporary insane ideas dependent thereon, angry, motiveless emotions, so that softening is often mistaken, temporarily at least, for dementia paralytica. This depends partly on the wide-spread disease of the vessels in the cortex, whereby the circulation and the nutrition of the cortex are essentially interfered with, and partly on the before-described, episodically occurring, inflammatory disturbances around the focus, every one of which causes an intense congestion with all its consequences. One must also keep in mind the rôle which the encephalitis accompanying softening plays in the above-mentioned hemiplegia and motor manifestations of irritation.

Prognosis.

a. Traumatic meningitis.—It is quite impossible to determine how far a meningitis can advance and still be capable of resolution; and just as little can the other question be answered, how far it must advance to surely cause the death of the individual. In point of fact, the post-mortem appearances in traumatic meningitis vary within such wide boundaries that the acceptation of still unknown, probably individual conditions can by no means be rejected. The quality of the decomposing products furnished by the external wound certainly has a most important influence; yet, as can be easily understood, accurate facts in this direction cannot be furnished.

Primary meningitis, produced under the influence of the putrefactive germs of the atmosphere, is one of the most dreaded complications of injury; we have already called attention to the advance, often very rapid, from a group of inflammatory symptoms indicating congestion only, to conditions of cerebral paralysis; if the functions of the brain are completely paralyzed, and the patient becomes comatose, the prognosis in primary meningitis cannot be other than fatal. The judgment, however, is materially affected in many cases by the uncertainty of the diagnosis. Who can to-day determine with certainty whether symptoms of irritation, occurring after injury to the skull, reaching a certain pitch, and then subsiding, really belong to a purulent meningitis or not? For the latter has no pathognomonic symptoms, as has already been sufficiently insisted upon, and we know as yet far too little that is certain about the accompanying febrile conditions to be able to form an opinion upon them.

There exists, without doubt, no sharp boundary between congestive hyperæmia and the transudations caused by it, with limited migration of formed elements of blood, and true, complete, purulent inflammation.

It is true that traumatic inflammation of the brain is not an absolutely fatal disease; on what its retrogression in certain cases depends is unknown; in the cases that have recovered, the degree and severity of the symptoms have differed, and it is impossible to say anything about the condition of the pia in those cases which, in spite of developed symptoms, have recovered.

b. Meningo-encephalitis.—In case of a superficial contusion of the brain the usual termination is acute suppuration, together with meningitis. This occurrence must always be looked upon as fatal. A complete cure, then, is possible only when diffuse meningitis does not take place, and when the pus in the brain finds a sufficient discharge through the open wound ; or when a passage is made for the pus by a primary trephining, or by the extraction of a loose splinter, or by the elevation of a broken fragment. Attention will be called later to the results of trephining made for this purpose (see the compilations of Bruns and Belmont, and the series of cases quoted in Bergmann, p. 294).

c. Suppurative encephalitis without accompanying meningitis, resulting from contusion of the brain, is certainly not capable of a spontaneous absorption ; at least thickening of the pus with gradual absorption and shrinking of the focus has not been observed with sufficient certainty. The ways in which a cure may nevertheless occur are spontaneous perforation externally, or artifical evacuation.

In spontaneous perforation externally, it is to be remarked that all the observations refer to chronic traumatic abscess, and that the cases in which an acute abscess perforated belong in the category of perforation through a hole or fissure caused by the injury, so that the appearance of the pus confirmed the diagnosis. In another series of cases an external abscess first formed, and only after its evacuation was the injury of the bone or the fissure discovered, which led several to trephine with different results.

A further series of acute abscesses of a traumatic nature finally pass into chronic abscess, and can remain during a very long period of latency without symptoms. To be sure, this will not essentially improve the prognosis of acute abscess, yet from this it appears that acute abscess of the brain is not an absolutely fatal disease.

d. Chronic traumatic abscess of the brain.—The greater part of abscesses belonging in this category end in death. A small number of cases have recovered spontaneously after perforation through the skull. The material in point is collected in Bruns (p. 976), and has received in recent times an addition through cases where the pus made an outlet for itself through the shattered roof of the orbit (Bauchet). Graeulich reports a case where, after a period of perfect latency lasting three weeks, brain symptoms appeared. A tense fluctuating tumor over the middle of the parietal bone was opened, and a fracture of the skull with depression was seen, through which pus escaped. In the cases cited by Bruns the following were the points of perforation: through the left, then through the right ear, finally out through the nose (Le Blanc); perforation through the left ear after thirteen years' latency, which was characterized by constant cephalalgia (Rust); perforation through the ear (Gama); perforation through the spongy lamina of the ethmoid (Schmucker); through the skull in the middle of the sagittal suture (M'Turk). These must count as very rare exceptional cases.

A second series of cases, ending in recovery, are those in which the pus was evacuated by fortunate trephining (Bergmann, p. 294). We shall mention these cases again under Treatment. The number of cases where the patient was saved is small; the number of those is tolerably great where the operation did not fail of its immediate effect, but in which death ultimately occurred. This should by no means stand in the way of an operation under favorable circumstances.

In spite of these successful cases, which unfortunately increase in number only very slowly, the prognosis of chronic traumatic abscess of the brain is to be designated as an unusually bad one.

e. Otorrhœal abscess of the brain.—Here, too, no cases are known of absorption and cure, with a focus-like residuum. On the contrary, a number of cases are known in which the abscess broke through the already diseased and, temporarily at least, permeable ear (Wilde, Lallemand, Cannstatt); and also some doubtful cases by Moos, Krukenberg, Sédillot, and post-mortem discovery of discharge of the collection of pus under the external skin. The number of these observations is, at all events, small; at least smaller than the number of those where a pyæmic affection with brain symptoms, which was interpreted as thrombosis of a sinus, got well spontaneously. Another mode of termination of acute abscess of the brain is by becoming chronic.

The chronic forms do not offer a better prognosis; they lead, without exception, after a longer or shorter time, to death. Indications of the possibility of evacuation externally are found;

for example, Wreden's patient, in whom, at the autopsy, the abscess was found to have perforated the squamous portion of the temporal bone, and discharged its pus under the temporal muscle, and Wendt's patient, in whom the abscess communicated through a hole in the frontal bone with the external subcutaneous abscess.

f. Abscess of the brain in pulmonary affections and other suppurations.—Nothing is known of cure or of promise of cure in this form of abscess; and the same is to be said of abscesses in pyæmia.

Nothing can be said concerning the prognosis of abscess following disease of the nasal mucous membrane and of other bones of the skull, on account of its great rarity.

g. Acute traumatic encephalitis without formation of pus. A number of anatomical residua and terminations of acute encephalitis have been briefly sketched above, and from it it appears that a relative cure of the so-called red softening is more frequent than that of other forms of encephalitis. Cases of recovery from undoubted traumatic encephalitis have, however, been collected by Bruns in his hand-book (p. 986), and it would not be difficult to increase their number. We ourselves have had undoubted cases of it.

Acute red softening in otorrhœa is so little known that we cannot form an opinion of the prognosis; at all events, it undergoes resolution much more rarely than traumatic encephalitis, and usually becomes purulent.

The prognosis of encephalitis around chronic abscesses, tumors, necroses, and apoplexies is essentially influenced by the nature and extent of the primary disease. In abscess and tumor, of course, the prospect is least favorable. In necrosis of every sort (worse in senile malacia than in embolus), and in apoplexy, the age and strength of the individual play a most important part. An encephalitic affection around a focus is, however, always a very serious complication; it often causes death by the consequent pressure on the brain (inflammatory œdema), or essentially impairs the prospect through increase of the focus, occurrence of new local and general symptoms, even if it should again subside.

Treatment.

It is not possible to discuss the treatment without going into surgery; and since we have no personal experiences here at our command, we are obliged to confine ourselves upon this point to the judgment of surgical colleagues (compare Bruns, Bergmann, p. 287).

The entire medical treatment in injuries to the head has one object in view, to guard against the usually fatal meningitis and encephalitis. For after this dangerous complication has fairly set in, there are very few cases in which life can be prolonged by confining the meningitis to a limited region, or where, under otherwise favorable conditions, an opening of the skull, when one is not already present, or an enlargement of the perforation, when it does not allow a sufficient discharge for the pus, can be thought of. Then there are pure traumatic abscesses of the brain whose existence and situation are indicated with so much certainty by the symptoms that an opening of the skull at the affected spot is not merely permissible, but is required. The therapeutic rules, then, might be arranged in the following categories:

Prophylactic measures.—It is a well-known fact that during the last decade these have become very much restricted. While in former times every fracture of the skull over which the skin was separated underwent operation, which was considered the chief guaranty against meningo-encephalitis, to-day we occupy an essentially different stand-point. To the old physicians, meningitis was not the infection of the pia, produced by the decomposition of the secretions of the wound, but a disease producing a certain quantity of pus, which caused pressure. This meningitis was to be anticipated by a free opening of the skull.

It is true that contusions of the brain were not well understood at that time, nor their relations to the symptoms and the subsequent suppurations. It was at a later period that an insight was first obtained into the action of the decomposing secretions of a wound on the tissues and the processes which take place when the access of the air is unimpeded. The reaction *against* early trephining became greater and greater, till finally Stromeyer, by his clear argument, swept away forever the reckless employment of the operation in all cases. As is usual in such cases, people again went too far in following Stromeyer; all trephining, even in perfectly evident contusion of the brain—indeed, each and every operation on depressed fragments—was shuddered at, notwithstanding the fact that Fischer taught us to know the dangers of pointed edges and splinters of bone.

With reference to treatment, we must first distinguish between fractures with and fractures without destruction of the soft parts.

In fractures with preservation of the skin, active interference is seldom called for; the normal process of repair is to be waited for, and the patient is to be brought under proper conditions. Depressed bone, over which the skin has remained entirely intact, should not be touched. For we know that the depression in itself, when it is not unusually large—which, indeed, can scarcely occur without injury to the external skin—is not the cause of the pressure on the brain, but in the great majority of cases the accompanying hemorrhage is. Even if the depression in an exceptional case has caused pressure on the brain, the most experienced surgeons are nevertheless of the opinion that it is better to wait for the brain to become accustomed to the pressure, and to let it stay—a risk which, it is proved, can be taken for a length of time without danger—rather than to call up the immediate danger of acute meningitis and encephalitis by an opening of the skull. For we do not know the condition of the dura and pia; perhaps they are just in that state which is the most favorable for the occurrence of diffuse inflammations, *i. e.*, they are lacerated and infiltrated with blood. But a contusion of the brain, a superficial mechanical destruction, may be present in such cases also. By elevating or trephining when the skin is not opened, the injured part cannot be saved; but if no air can gain access, the process of repair is unaccompanied by danger, at least in the majority of cases. If, however, we let the air come into contact with the contused brain, an acute infection of the pia by the most powerful exciters of inflammation is no longer to be prevented. But the exclusion of air is also a *sine* *qua non* for the innocuity of the processes of repair and retrogression in contused portions of brain. There are always cases enough, however, where, in spite of a closed skull, the contusion of the brain is transformed into abscess.

The question is, what shall be done when, in a fracture in the temporo-parietal region, hemorrhage from the middle meningeal artery can be surely diagnosticated from the symptoms (appearance of positive symptoms of pressure some time after the injury, hemiparesis)? We know that large extravasations of that sort, under expectant treatment, have finally ended in absorption and recovery. In certain cases the occurrence of secondary hemorrhage can be positively recognized, but the presence of hemiparesis gives no idea of the condition of the pia and the superficial parts of the brain. Trephining under such circumstances, in order to secure the bleeding artery and stop the hemorrhage, may certainly serve the latter purpose ; it has already done so ; but access of the air to the meninges is thus permitted, and the danger to the patient greatly increased thereby. Meningitis is unavoidable when the dura and pia are contused and infiltrated with blood. Without the entrance of air, there was yet a chance, even if the extravasation became larger; for symptoms of pressure, occurring some time after the injury and lasting for a long time, can disappear spontaneously.

As soon as the skin is torn over a fracture, then, of course, all precautionary measures, the object of which is to prevent the access of air, are useless. It makes no difference also whether the wound of the skin leads directly to the fracture, or whether periosteum and connective tissue still cover it.

In exposed fractures, without depression, it is evident that the question of trephining will not force itself into the foreground. On the contrary, if the fracture is only a fissure of moderate extent, and if the edges of the wound are not contused, it may be closed with sutures, with the hope of a favorable result.

Bergmann recommends this treatment, and gives examples where fractures so treated (when the condition of the soft parts otherwise allowed of it) healed like simple fractures, and the wound in the skin healed *per primam*, so to speak.

The case is quite different in exposed fractures, with depression, for then it can do no harm [the contused endocranial parts being already open to the entrance of air], but, on the contrary, is of great advantage, to raise or saw off the depressed fragments, for we are sufficiently acquainted with their action in the opened skull.

Surgeons are not yet agreed as to the proper treatment. One party still think that energetic operative treatment of such wounds is required ; others hold to the opposite extreme, and will hear nothing of early trephining and extraction of splinters, while a third set occupy the middle ground, and make the treatment depend on the circumstances of each case. This fact appears to prove that in the subject itself—that is, in the knowledge of the different courses pursued by these wounds, and especially in the estimate of the results of the operations—the necessary clearness is not yet attained. One should read the excellent remarks of Bergmann upon this point. He finds the reasons for this difference of opinion in the following grounds:

1. It is true that trephining removes the dangers arising from the presence of splintered fragments, but it involves a large opening and large, exposed surfaces of bone and soft parts. This is possibly a worse condition than the existence of a few broken fragments projecting inwards, when the external opening, as is often the case, is small, and leads in an oblique direction to the fracture.

The last circumstance gives to many injuries of that sort much more the character of a subcutaneous fracture ; and, when the relations are otherwise favorable, the latter situation is, without doubt, to be preferred to an open trephining wound. This shows already that one principle is not to be carried out in all wounds of this kind.

2. Nothing certain can be got from the statistics which thus far have been collected, because the work has not been properly done ; all cases have been thrown in together, without separating the heterogeneous ones, and comparing only the similar ones.

3. The rules which have been given for the performance of trephining, or its omission, are found by Bergmann to be quite as impracticable. Depressions were divided into superficial and

deep, and those over a quarter of an inch deep (Bruns) belonged to the last. The rule was to trephine immediately in depression with accompanying symptoms, and not to trephine in depression unaccompanied by the signs characteristic of a focus. This cannot be accepted, for it is very seldom that the brain symptoms depend on the depression itself, consequently the elevation or trephining can do no good, and may make things much worse. And as for the brain symptoms, they are the result of a contusion of the brain or of an extravasation of blood; the former cannot be in the least improved by trephining and elevation; and as for the latter, we have already stated briefly what is necessary to be done in hemorrhage from the arteria meningea media.

In this manner the field of early operations and early trephining, in cases of injury to the bones of the skull, is essentially restricted; there exists really no other rational indications for such operations than that designated with great accuracy by Fischer; splinters and foreign bodies, in cases which have a certain resemblance already to a trephining wound, or at least which possess its unfavorable characteristics, must be removed. Therefore early operations of this sort, according to our present knowledge, should only be made on splinterings of small extent, which vary in size but little from that of the trephining wound, and where an examination shows a splintering inwards, a displacement and wedging of the particles under the edge of fracture. Simple extraction of the fragments and elevation is not always sufficient; in some cases the fragments are wedged in so firmly that early trephining is necessary. The result of this is a wound less unfavorable for meningitis than the primary injury was. A description of the special rules for such manipulations we must leave to surgery. In practice the question is by no means so simple as one would, *à priori*, suppose; for example, the doubt will sometimes arise whether it is not better to leave this or that small splinter of bone behind, because it is impossible to determine in a small field of operation whether the dura is torn or not, or to what extent it is. It is certainly better to leave a fragment fast in the dura than to withdraw it forcibly.

In the large majority of cases of depression with external wound, the treatment, therefore, according to what has been said, should be expectant; for only a small number of wounds correspond to the provisions mentioned as requiring a primary operation. As for the further treatment, we stand with those who consider the open method of treating the wound with strong antiphlogistic measures as that which leads most easily to the desired result.

The question now is, whether the course which has been pictured to this point, and which depends on the conditions of the bones, essentially changes as soon as more or less distinct general or local symptoms on the side of the brain are joined with it?

The knowledge of an injury to the brain cannot alter the above principle; on the contrary, the recognition of an injury to the brain can only serve to strengthen it.

If general symptoms appear after an injury without exposure of the bone, the question of operative treatment again comes up. Without doubt the treatment would by no means be confined within its actual narrow limits, if those pictures of contusion and compression of the brain really should exist which the old surgery represented as the only ones. But, as compared with combinations of different sets of symptoms, they seldom are found uncomplicated. *Since, for this reason, an absolute diagnosis of the cause can never be made in ordinary cases of pressure on the brain; since, even when the locality of a hemorrhage can be accurately determined, we are not able to exclude absolutely the existence of contusion of the meninges and superficial destruction of the brain; and since a hemorrhage, supposed to be between the dura and the bone, may prove to be one between the dura and the surface of the brain, with tearing of the pia and lesion of the brain; since we know that symptoms of pressure in the brain, of moderate intensity, may be transitory; since, finally, the forcible removal of anything pressing on the brain creates an opening in the skull which directly leads to diffuse meningitis if the pia and surface of the brain are lacerated, therefore, in the majority of cases, operative interference must be abstained from.* It can be a question of operation probably only in one of those perfectly plain cases where a hemorrhage from the arteria meningea media is beyond a doubt;

that is, when symptoms of pressure first appear some time after the injury, and steadily increase up to a certain pitch. The symptoms (unilateral paresis) and the nature of the injury must, at all events, confirm the diagnosis. In such cases trephining, to stop hemorrhage from the arteria meningea media, is allowable, and it has been performed several times with good result. It has been made at the seat of injury and the proposition has been made (Vogt) to trephine at the point of election (at the crossing of two lines, one of which runs horizontally two finger-breadths above the zygomatic process, the other vertically, a finger's breadth behind the ascending spheno-frontal branch of the malar bone). Adams and more recently Bergmann reject this method of operation ; the latter thinks—and properly—that the operation should be confined to the plainest and simplest typical cases, and advises that if these symptoms are in the least indistinct, the protecting covering of the brain should be preserved.

Since, now, in following these different indications, we find ourselves finally, in the majority of acute cases, in presence of partial pressure on the brain, with or without a wound of the skin and of the bone, for which we have decided not to operate, the question presents itself, what measures we shall adopt against the subsequent symptoms, namely, the congestions, which transform the partial into a general pressure on the brain, and the meningitis and acute encephalitis. Unfortunately they are very limited.

When the pressure on the brain has become general and has arrested the passage of the blood through the cortex, venesection by diminishing the amount of blood in the body, will undoubtedly re-establish circulation within the skull.

1. A free escape for the stagnating blood in the skull is thereby furnished, and this in turn allows the re-entrance of the arterial blood into the cortex of the cerebrum. When, therefore, with congestion and symptoms of pressure, the pulse is full, and the power of the heart is undiminished, venesection should not be delayed, and it may even be repeated several times in strong constitutions. If, however, this quality of the pulse is not present, if the symptoms are rather those of commotio cerebri, if the pulse is small and weak, then venesection, at that time at

least, is strongly to be deprecated. The slow, large, full pulse, associated with general symptoms and disturbances of respiration, is then the chief indication for bloodletting. Local depletions have for obvious reasons lost credit, especially with military surgeons, and physicians, too, are gradually coming to the same opinion, because their action seems not to be energetic enough. In practice among children they will never, indeed, lose their value.

2. The application of ice, to produce contraction of the arteries and, as a result, increased resistance against the pressure of the blood; and, secondly, as an antiseptic to restrain as much as possible primary decomposition in an external wound. The application of the ice-bag is never to be omitted when there is danger of excessive reaction about a wound, or when there are signs of congestion. Irrigation with ice-water has been often substituted, and apparently with advantage, for the application of ice (Pirogoff, Stromeyer, Bergmann, see his hand-book, p. 205). We have found in such cases, that pouring pretty large amounts of ice-water repeatedly upon the patient's head, while he sat in a tepid bath, had an excellent effect.

3. The administration of drastic cathartics with the view of stimulating as much as possible the absorption of fluid from the lymph-spaces. Those best adapted to our purpose are the vegetable ones, senna, jalap, colocynth; the addition of the favorite drug calomel, as long as it is regarded as a simple aperient, is allowable.

4. It is difficult for those whose experience in surgery is limited to convince themselves of the efficacy of specific means in the traumatic form of meningitis and encephalitis. If it were not for the renowned Stromeyer, who spoke so energetically in favor of mercury, we would here join those who expect rather an aggravation than an improvement from the use of mercury pushed to salivation. Since, however, this method of treatment is being constantly recommended again by surgeons, it should by no means disappear from our list of remedies. The inunction of mercurial ointment, which can be supplemented by small internal doses of calomel, acts quickly. The action sought is the commencement of salivation. An essential obstacle to the suc-

cess of this action is that the patient very frequently dies before it can be obtained.

Following the advice of sound surgeons, we have not entirely discarded primary trephining, but it should be confined to few cases. Very different has been the experience of operations performed, not while the wound was fresh, but during the reactive processes of the first few days—especially inflammation of the intra-cranial organs.

Intermediate trephining and other operations in perforating wounds of the skull are to-day almost universally condemned; any disturbance of the parts affected with inflammation can, in fact, only increase the production of deleterious secretions from the wound; the cause of diffuse meningitis does not lie in a collection of pus, which is capable of removal, but in an infiltration of pus into the pia, which no trephining and elevation can remove, but which can be made essentially more acute by the increased inflammation.

Therefore, only one indication for trephining remains, where we can express ourselves positively and without essential limitation: *it may be made for abscesses of the brain the diagnosis of which is certain, and the location of which is known beyond a doubt.* Concerning the occurrence of these conditions, we refer to what has already been said; unfortunately the number of abscesses of the brain will always be small, in which the symptoms will be so clear that the trephine can be boldly put on with the conviction that the focus will be immediately found. There is yet another danger; in some cases, after making the hole with the trephine, the dura has been found intact, and the question has then arisen whether the existing circumstances justified its division (compare Bergmann, p. 295, where the cases in point are collected). Some surgeons have even hazarded an incision deep into the brain without attaining their purpose thereby; for in some cases the patient nevertheless died after it, and in others the abscess was not found. Unfortunately it is now scarcely to be supposed that a much greater certainty in the diagnosis of the locality of the abscess will be attained than is the case to-day, and, therefore, trephining will always be confined to those cases where the isolation of the symptoms of localized disease, existing aphasia and the like, furnish a sure starting-point. The situation is much more favorable when an open hole or fissure in the bone lets the pus ooze out, for then there can be no mistake as to the locality.

Renz succeeded in such a case (Theodor von Renz, Mittheilungen aus der Praxis), after extracting the blade of a knife, in emptying the abscess, which lay deep in the brain, by successive introductions of a subcutaneous syringe, and cured the patient; the same patient lived eight and a half years free from all brain symptoms and died from hemorrhage of the lungs (or stomach?). To attain a constant and equable traction Renz had an aspirator purposely constructed (loc. cit. p. 183).

Abscess of the brain from otorrhœa is scarcely ever accessible to surgical treatment, as follows directly from the fact that the greatest number of these abscesses occupy either the temporal lobe or the cerebellum. In such cases, therefore, even if the symptoms are of an unequivocal nature, we are thrown back on the usual symptomatic treatment. The only possible help which can be given to the patient is the best possible regulation of the circulation in the skull by favoring the flow through the veins, the application of cold, and the appropriate use of narcotics.

Abscesses of the brain in pulmonary affections, in purulent processes in the body, as well as pyæmic abscesses, are scarcely accessible to treatment; the position taken by the physician will be that already often described.

The acute encephalitic diseases unfortunately fall under mere general categories in treatment. The general rules applicable whenever any form of pressure is exerted on the brain are the only ones at our command. We consider it superfluous to repeat the special directions here. The treatment will, according to general laws, sometimes have as its object to assist venous discharge from the brain by emptying the peripheral veins of the head or of vessels farther removed, also to act on the cerebral vessels by energetic application of cold; at other times especially prominent symptoms of a sensory or motor nature will give a symptomatic indication. In this category belongs the treatment of severe convulsions by the judicious exhibition of narcotics, either in the usual way, or subcutaneously, or by enemata. Conditions of high fever, when the circumstances otherwise allow it, demand the application of the known antifebrile remedies; rapidly progressive paralysis of the brain and sudden collapse require a vigorous use of stimulants. The results depend entirely upon the circumstances under which the encephalitis arose, and it is evident, therefore, that in the majority of cases the diagnostician has more opportunity to distinguish himself than the *therapeutist*.

HYPERTROPHY

AND

ATROPHY OF THE BRAIN.

HITZIG.

HYPERTROPHY OF THE BRAIN.

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History.

Morgagni was one of the first to notice that in certain autopsies the brain was apparently too large for the skull, for it was not possible to replace the cerebral mass within its bony envelope after this had once been removed. Laënnec subsequently found a similar condition of the organ, with flattening of the convolutions, in patients in whom he had, as it afterward appeared, erroneously made a diagnosis of hydrocephalus. By the publication of a remarkable case, Scoutetten then attracted general attention to this disease. After a time, during which a series of cases had been published, Virchow claimed that a distinction should be made between the increase (hyperplasia) of the actual nervous substance and that of the supporting tissue or framework (neuroglia). The former he referred to the department of physiological changes, while the latter may with certainty be considered pathological.

Classification.

The name, hypertrophy of the brain, has hitherto been made to include a series of different, but as it seems exceptionally rare pathological processes. These may be divided, according to their locality, into general and partial hypertrophy of the organ.

In general hypertrophy we must again make a distinction between the acute and the chronic form, inasmuch as they differ in the combination of symptoms and the danger to life which accompany them. With these forms it is customary also to consider new formations of cerebral substance within the cerebral envelopes (heterotopia).

Etiology.

So far as conclusions can be drawn from the rather scanty material on hand, it would seem that in most of the cases under consideration there was an original defect of development, which might depend upon a *hereditary* or only upon a *congenital neuro-pathological diathesis.* In this connection a comparison with cases of *peripheral* multiple neuroma is instructive, since in both there is a tendency to abnormal development of nerve-tissue, and sometimes, indeed, both conditions seem to be combined. Thus, Hesselbach[1] mentions the case of a man, aged thirty-nine years, who, besides symmetrical swelling of the peripheral nerves and the formation of knots on them, had also a considerable enlargement of the sympathetic ganglia and of one middle cerebellar peduncle. As this man had inherited his disease from his father, so had two others, brother and sister, mentioned by Hitchcock,[2] inherited their neuromata from their mother. Betz also reports the case of a family in which the large heads of the ancestors were followed by cerebral hypertrophy in several of the descendants. Both these conditions, the peripheral and the central excess of nervous tissue, are likewise a frequent accompaniment of *idiocy*, while in other cases at least a marked delay in intellectual development is noticed.

A further proof of the congenital cause of cerebral hypertrophy is to be found in the circumstance that it is noticeable in much the larger proportion of cases either immediately after birth or in the earliest years of life. The greater part, although not all, of those cases in which it is asserted that hypertrophy was developed in more advanced years, are not very well authenticated. The majority of Sims' observations, in particular, are of little or no value, for they have evidently not been subjected to any critical analysis.

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[1] Beschreibung der path. Präparate, welche in der k. anatom. Anstalt zu Würzburg aufbewahrt werden. Giessen, 1824, S. 284, 362.

[2] American Journ. of Med. Sci., 1862, April. Copious bibliography of neuroma in *Virchow's* Geschwülste. III. Bd. S. 233.

In a case reported by Tuke a traumatic injury seems to have been the cause. It is the case of an idiotic man, thirty-seven years of age, who received a blow upon his head when ten days old, and had from that time suffered from spasms. Dance likewise reports a case of a patient, previously healthy, who had cerebral symptoms immediately after an injury to the head, in his fifteenth year, and who died when twenty-seven years old. *Lead* has also been mentioned as a cause by some. Thus, Andral found the characteristic *post-mortem* appearances in two white-lead workers, who were sick only twenty-four hours, and died with the characteristic symptoms of eclampsia saturnina. Mériadec Laënnec, Bright, and Papavoine mention similar experiences.

According to Andral, frequently repeated congestion of the brain may give rise to hypertrophy. If it be remembered, however, what a very large number of persons, in one way or another, are exposed to these vascular changes, and yet how rare our disease is, we may be permitted to assign to this condition a purely hypothetical significance. Even in Andral's writings this view is only advanced as conjectural, and later authors make still less of it. On the other hand, we would not, of course, deny the possibility of an increased supply of nutritive material being brought to the parts during the progress of hypertrophy. But this would then appear to be the consequence, and not the cause of the disease.

Pathology.

Anatomical Changes.

The anatomical changes naturally differ very greatly, according as the hypertrophy is partial or general, and there are marked differences even between cases of sudden and those of gradual increase of the organ.

If general hypertrophy begins in the earlier years of childhood, the *skull* follows the abnormal development of its contents, and the external form of the head, similar in all respects to that of hydrocephalus, justifies a conclusion in regard to the amount of the increase, if not as to its nature. Virchow has proposed the name "cephalo "(cephalones) for this form of head, which so closely resembles hydrocephalus, but does not contain an abnormal amount of water. Rokitansky says: "In certain cases, with rapid and great development of the disease, there occurs in the infant's skull a loosening, a diastasis of the sutures of the cranium, with red coloration, or suffusion of the articular cartilages." If the disease makes its first appearance when the skull has become compacted, there is found, instead of enlargement of the skull, a general or partial atrophy of its individual bones, resulting from compression, many translucent spots and roughnesses appearing on their tabula vitrea. This atrophy of the cranial bones was also noticed on the affected side in Tuke's case of unilateral hypertrophy.

The *cerebral membranes* are, almost without exception, compressed against each other and against the bones by the viscus, so that the dura mater adheres firmly to the skull; they are thinned, the blood-vessels scarcely visible, and every trace of cerebral fluid is lacking in the inter-meningeal space and in the meshes of the pia mater.

The *lateral ventricles* are also so compressed that they either contain no fluid or only a very small amount. When, notwithstanding this, Sims claims to have found an unusual collection of water in many of his autopsies, his statements are to be received with the greatest distrust. In these cases the secretory pressure would have had to be still greater than the pressure of the hypertrophied cerebral substance.

It is stated by many observers that, immediately after removal of the calvarium, the brain, still covered by the dura mater, has projected so that it has been scarcely possible to replace it within the skull. But almost always the convolutions have been flattened and so pressed together that the sulci seemed entirely obliterated. The brain-substance shows a marked change of consistency; it is tough like boiled white of egg, cheese, or rubber. Dance and Andral could draw it out like rubber without its breaking. Tuke made no impression on it with a column of water five feet high.

The increase in consistency cannot be referred *entirely* to changes of structure. I have shown elsewhere[1] that the extra-ventricular fluid is absorbed by the cerebral substance soon after death. In hypertrophy of the brain, besides the absence of this, which acts as a factor for diminishing the consistency, a large part of the normal contents of the vessels whose serum would produce maceration is also wanting. For these reasons the brain seems more consistent, just as it does in animals which I have killed by separating the heart from the large vessels, and immediately examined. Aside from this, the changes occurring in the cerebral tissues still remain to be considered.

Steinberg invented an instrument for measuring directly the consistency of the brain, which has not yet been further tested.

As a rule there is excessive anæmia in the organ. Scarcely any divided vessels are to be seen; the gray substance, as well that of the convolutions as that of the central ganglia, is so pale that it differs but little from the white substance. However, this extreme anæmia of the viscus and its soft envelopes is probably the result of the final compression; that is, if the patient died of the cerebral hypertrophy itself and not of some intercurrent disease. In that case we should expect to find this anæmia only in acute forms of the disease. In this respect the case of Scoutetten is especially instructive. A child five and a half years old, with a head large enough for a robust adult, died of an intercurrent disease, and not only was the cerebral substance found of a rosy color, but the pia mater also was unusually hyperæmic; nothing is said about flattening of the convolutions, but the other signs of hypertrophy were present. Hence, it is quite possible that in other cases there is also hyperæmia, excepting during the last days of life—the anæmia occurring only as the result of an increase of bulk, which finally cannot be compensated in any direction, and so becomes only the final cause of death.

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[1] Ueber den Ort der extraventriculären Cerebralflüssigkeit, Reichert's und du Bois Reymond's Archiv. 1874, H. 3.

In judging of the statements on hand concerning the *increase of the entire weight* of the brain, we become painfully aware of the lack of any statistics with regard to the weight of healthy brains, based on a systematic method of investigation and on a sufficient amount of material.

We possess indeed a very large number of observations upon the weight of the whole brain as well as of its separate portions; yet different works on the subject contain a disproportionate number of incompatible measurements. Not only has the division, from each other, of individual portions of the central nervous system been made according to different methods, but, what is much worse, healthy and diseased brains are included in the same tables, so that it is impossible to judge in how far the pathological increase or decrease in the size of the organ or in its specific gravity influences the average results given. Finally, but few, even of the later authors, have paid any regard to the proportion existing between the brain and the size of the body. It is clear that only a proportion derived from both factors can be used for our purpose, for the normal brain of a person six feet tall would clearly seem hypertrophic for a person only five feet tall. The correctness of this view, and the incorrectness of so many conclusions that have been arrived at, is seen from the comparison of the weight of the brain and body in the two sexes. It was known even to Aristotle that the female brain was lighter than the male, and this fact, of all that are here discussed, is almost the only one about which no difference of opinion has arisen since. According to Huschke[1] the difference varies, according to age, between 82 and 192 grammes (ʒ xxss. and ℥ vi.); according to Meynert,[2] the difference is 126 grammes (℥ iv.); and according to Weissbach,[3] 159 grammes (℥ v.).

But if we likewise take into account the measurements of Tiedemann,[4] who included the weight of the body in his observations, then this anatomical fact loses its unpleasant aspect. For the relative weight of the brain to that of the entire body proves to be in favor of the female sex. In woman the brain weighs from one forty-fourth to one-fortieth as much as the body, in man from one forty-second to one forty-first. The same thing holds good with regard to the absolute weight of brain of Frenchmen, which is decidedly less than that of the Germanic race. The fact is, the entire bodily weight of our neighbors is less than our own. According to Hushcke, the brain of the adult forms on an average somewhat more than two per cent. of the weight of the body. It seems to me, indeed, that all these observations, especially those which relate to the weight of separate parts of the brain, are of value only if on every occasion not alone the height of the body, but also its weight and previous state of health are noticed. On the one hand, although not proven, it is yet very possible that in the original plan of development powerful muscular formations go hand in hand with a similarly powerful development of the central organs. On the other hand, this relation may doubtless be greatly influenced in one direction or another by disease. Under these circumstances it would be of great service if physicians, occupying official positions (such as coroners, police surgeons, etc.), would turn their attention to obtaining statistics with regard to the normal weight of the brain; for they are the only men who have the opportunity of observing the weight of the brain in a large number of persons who have died suddenly while in good health. The importance of these considerations becomes evident when special tables of weight are considered. The clearer proof of this is given by table three of Sims, arranged according to age. In it we find, for example, that the heaviest brain of the second class (two to three years), weighing 1162.2 grammes (three pounds one ounce), was heavier than the lightest brains of all others, with the exception of that of the seventh class (fifteen to twenty years), which weighed 1218.9 grammes (three pounds three ounces). The heaviest brain between four and five years, 1275.6 grammes, however, exceeds the latter by 56.7 grammes. A number of similar paradoxes may be discovered.

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[1] Schädel, Hirn und Seele. Jena, 1854, p. 60.

[2] Das Gesammtgewicht und die Theilgewichte des Gehirns u. s. w. Vierteljahrschr. f. Psychiatrie. 1867, I. 2. p. 126–170.

[3] Die Gewichtsverhältnisse der Gehirne oesterreichischer Völker u. s. w. Arch. f. Anthropologie. I. p. 191–218 and 285–319.

[4] Das Gehirn des Europäers mit dem des Negers und Orang-Utangs verglichen. Heidelberg, 1837.

The very complete compilations and weighings by Huschke show that during the course of the thirtieth year the brain reaches its greatest weight, this being on an average in man 1424, in woman 1272 grammes. Of this about fifty grammes are to be referred to the pia mater and the arterial network. Weisbach, however, places the greatest weight between the twentieth and the twenty-ninth years. Meynert, who, to be sure, weighed only the brains of the insane, found the average weight in men between twenty and sixty-nine years old, to be 1296, in women 1170 grammes. According to him the greatest weight is attained in men between the age of thirty and forty, and in women not until between forty and fifty.

The following data, concerning the average weight at different periods of life, we take from Mauthner to the end of the first year; from Sims to the end of the tenth year; and for the later years from Huschke: in all cases only the average is given.

10 days—11 Weeks.	12—20 Weeks.	6—12 Months.
(13½ Austrian ounces) 337 grms (?).	(16¾) 420 grms.	(20½) 512 grms.

1—2	2—3.	3—4.	4—5.	5—10.
935 grms.	1030 grms.	1083 grms.	1105 grms.	1146 grms.

	10—19	20—29	30—39	40—49	50—59	60—69	70—79	80—90
Men.......	1411	1419	1424	1406	1398	1291	1254	1303
Women....	1219	1260	1272	1272	1239	1219	1129	1186

But, for forming a judgment in regard to a possible hypertrophy, it is not sufficient to know the average weight only; it is also essential to know the maximum weight. From the tables given by Huschke, this would range from 1500 to 1600 grammes in adults. On the other hand, such remarkably heavy brains are ascribed in literature to prominent persons, that the figures alone would incline one to doubt as to the correctness of the statements, even if it were not known that the skull of Cromwell, preserved at Oxford, which is supposed to have held a brain weighing 2233 grammes, is by no means remarkable for size. Lord Byron's brain is said to have weighed 2238, and that of Cuvier 1829 grammes.

Such data justify the suspicion that the scales of the investigator in these cases played a less important part than a preconceived idea of his mind, the general correctness of which, apart from the details of the individual case, still remains to be proved. It must be granted that the individual genera, and even the species in the animal kingdom, gain in relative weight of brain with their psychical development. But there is nothing in this to justify the common supposition that, within the species, the more richly endowed individuals must have absolutely heavier brains. It is much more likely that Galen was right when he stated that the quality was more important than the quantity. In fact, the largest heads are by no means found upon the shoulders of the most important men. But if we enter upon a psychological analysis of that which from all time has made men celebrated, and which will none the less serve as the future measure for human greatness, it is evident that it implies nothing for which organs are needed which shall vary much from the average human organs. Aggressive, fruitful ideas, and the wealth of action flowing from them, have some community with wit. Here as there the peculiarity of the performance is not in the fact that something is advanced which an average or a well-educated mind cannot perceive or meditate upon, because the corresponding organ is not granted to it, but rather in the ability to take generally received perceptions and ideas, and to arrange them in an original manner, to place them over against one another, and to unite them in such a way that out of them arises something that seems absolutely new, nearer to perfection.

It would be well if, with the rest of the crude teachings of Gall, his ideas in regard to localization should gradually be discarded.

In view of all these circumstances it becomes evident that, in the present state of our knowledge, the absolute weight of the brain, considered without reference to its other relations, justifies the diagnosis of hypertrophy only when the average weight is exceeded in an *extraordinary degree.* This was the case in some of the four brains weighed by Virchow.[1] In a three year old child the brain weighed 1911 grammes; in a thirteen year old lad, 1732.5; in a twenty-three year old girl, 1675; and in a person fifty-four years old, 1872.5 grammes.

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[1] Untersuchungen über den Schädelgrund.

The *specific gravity* of the cerebral mass should also be taken into account in these estimates, more than has been done heretofore. Tuke found it unchanged, on the diseased side, in his case of unilateral hypertrophy; it was 1036 on both sides, differing from the results obtained with normal brains in the fact that it was the same in the gray as in the white substance.

Lately the specific gravity has been calculated by Charlton Bastian[1] and by W. Krause and L. Fischer.[2] According to them the specific gravity of the gray substance seems, on the average, to be about 1031, and that of the white substance between 1036 (Krause and Fischer) and 1040 (Bastian).

As a rule, only the cerebrum is affected with so-called hypertrophy. Yet there are a few cases—for example, that by Sweatmann—in which the cerebellum has also been said to be affected.

Virchow's declaration, that in cases of pathological hypertrophy the *increase of bulk* is especially *in the neuroglia*, was tested in two cases, by Tuke and by Landouzy. The first investigator found, indeed—especially in the posterior lobe of the diseased hemisphere—a very marked increase of connective tissue, which he has illustrated by drawings; there was also an atrophic condition in the cellular layers of the cortex, which might have had its origin in a concomitant inflammation of both cerebral membranes. In Landouzy's case, on the contrary, Magnan could find absolutely nothing abnormal on microscopic examination.

The changes found in *partial hypertrophy* are yet more rare, and, in part, of very doubtful nature. Except the case by Hesselbach, already mentioned, and Tuke's, which has likewise been mentioned repeatedly, I find similar accounts given only by Sims and Sangalli. The first reports that in a woman, sixty years old, who had suffered for two years from cerebral disease, the left hemisphere occupied two-thirds of the cranial cavity, the left corpus striatum also being of double size. In an insane patient, sixty years old, with hydrocephalus externus, the right corpus striatum, the left thalamus, and the pons Varolii, were much increased in size. Sangalli's three patients had not suffered from nervous symptoms; in one the left thalamus was 47 mm. (1.8 inches) long and 27 mm. (1 inch) broad, while the corresponding dimensions of the right were only 37 mm. (1.4 inches) and 18½ mm. (.64 of an inch); it was composed of nervous substance. A second case consisted in proliferation of the cortex at the base of the brain, the essential character of which seems doubtful. In a third case there was a growth of the ventricular surface of the right corpus striatum of the size of a bean. (Glioma ?)

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[1] On the specific gravity of different parts of the human brain. Journal of Mental Science, Jan. 1, 1874.

[2] Henle und Pfeufer's Zeitschr., xxvi. D. 306–331. The older bibliography in Huschke.

Symptomatology.

It has already been mentioned that the features of the disease are quite different, according as it is a case of acute or chronic development. Virchow describes a classical case of acute hypertrophy.[1]

A previously healthy woman, twenty-four years old—a prostitute—was suddenly attacked with gastric symptoms, abdominal pain, retention of urine, severe headache, great weakness and insensibility of the limbs. On the second day after she came under observation—the fifth of the disease—there was also vertigo, somnolence, nocturnal delirium, inability to stand, and a pulse of 44. The patient died on the eleventh day, without any marked change in her condition, the pulse rising to 70 or 80, with dyspnœa and dysphagia. Agreeably to the statements of most authors (excepting Rokitansky), the medulla oblongata was perfectly normal in this case.

Headache almost always plays a prominent part, as in the case just mentioned; it often has intermissions and remissions, but generally is fearfully severe. Next *spasmodic attacks*, of an epileptic or epileptoid nature, occur; though Mér. Laënnec goes too far in saying that they are a constant symptom. Sometimes there are partial spasms or tremor. The retardation of the pulse is likewise by no means constant, but occasionally there is an increase, even to 152, as in a case by Steiner and Neureuther.

Still less can be said positively in regard to the symptoms of *chronic cerebral hypertrophy.* The condition of the intelligence would be especially interesting. But even herein the statements vary. In a number of cases the patients—all children—either showed premature mental development, as in Elliotson's case,[1] or at least a degree of development corresponding to their age. In other cases again there was more or less weakness of mind, amounting even to the highest degree of idiocy. A similar criticism may be made in regard to the sleepiness and greediness of patients, which is sometimes mentioned. Some children, on account of the excessive weight of their head, frequently fall forward to the ground.

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[1] Ueber die Natur der constitutionell-syphilitischen Affectionen.

Excepting the headache already mentioned, disturbances in the nerves of general or special sensation are comparatively rare. The optic nerve, in particular, is very seldom mentioned. Steiner and Neureuther alone speak of the sudden occurrence of blindness ; others merely mention photophobia. Possibly a careful ophthalmoscopic examination of the retina would give more positive results. Tinnitus and subjective noises in the head are often present.

Course.

It may be a question whether the course of the disease, even in those cases which appear to be primarily acute, may not actually have been protracted, the disease not causing trouble until the space in the cranium becomes limited. This view was held by Andral, and is accepted by Hasse. There are no data upon which to found an opinion.

Many chronic cases, which are either already marked by large heads, or show other cerebral symptoms (headache), finally take on an acute character and end quickly in death.

Not rarely the fatal termination occurs only in consequence of an apparently insignificant intercurrent affection—as a diarrhœa or a bronchitis. The cause is to be found in the total revolution in the relations of the circulation within the cranial cavity. A slight amount of hyperæmia increases the pressure beyond the limits within which life is possible; a slight increase of the possibly already existing anæmia leads to fatal diminution in the supply of nutritive material.

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[1] Medical Gazette, 1832. Vol. IX., p. 622.

Chronic cases may extend over many years, and, as it seems, remain stationary. A sudden increase in volume, whether in a brain previously healthy, or in one already chronically enlarged, may cause very rapid death, as on the eleventh day in the case quoted from Virchow.

As a brief summary of what has been stated, we may say that *acute hypertrophy* appears and runs its course with the symptoms which are common to all diseases that quickly restrict the space allotted to the brain, while the *chronic* form, especially in children, cannot be distinguished from chronic hydrocephalus.

Diagnosis, Prognosis, Treatment.

It must already be evident that the wisest plan would be not to make the diagnosis of cerebral hypertrophy until the autopsy, although it is certainly well during life to bear in mind the possibility of the disease. Punctures and injections of iodine are still practised on hydrocephalic heads even at the present day, and good results are claimed for them. It would certainly be disagreeable if this procedure, hazardous at best, should be undertaken on a hypertrophied brain.

On account of the impossibility of making a diagnosis, a prognosis cannot well be given, but it may be deduced from what has been said. There is of course nothing to be said upon treatment.

Heterotopia of Brain Substance.[1]

This anomaly, first described by Virchow,[2] and hitherto principally of pathologico-anatomical interest, is closely allied to partial hypertrophy of the brain. Encephalocele (hernia cerebri vera) forms a teratological transition between the two, and at the same time constitutes an exquisite form of heterotopical development, inasmuch as, according to Virchow,[3] this condition sometimes consists merely of the presence of superfluous and extra-cranial masses of cerebral substance, without any coincident ventricular dropsy. Here belong a number of very valuable observations made by Simon. Small accessory gyri, from the size of a millet-seed to that of a pea, were found situated on the summit of the convolutions. Section showed that a thin line of white medullary substance passed through the normal cortex, entered the accessory cortex, and then spread itself out in a fan shape.

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[1] Of course we do not here consider the new formation of cerebral substance in other parts, as in ovarian tumors.

[2] Gesammelte Abhandlg. Berlin, 1862. 2. Aufl. S. 898.

[3] Geschwülste. Bd. III. S. 270 et seq.

Virchow himself describes in one instance an apparently new formation of gyri within the white substance of the posterior lobe. (It may be said, in passing, that this is the most interesting of the cases at present on record.) In one spot the heterotopic mass of gray substance was connected with the cortex, but elsewhere it appeared as stripes of gray substance, situated around a central stripe of white matter, the gray matter sometimes running its course continuously and sometimes being interrupted by bands of white matter. The central stripe of medullary substance resembled the medullary substance of the convolutions, both as to its blood-vessels and as to its nerve-distribution.

Virchow found similar hyperplasiæ, likewise in connection with gray substance, in the form of hyperplastic malformations of the cauda corporis striati. The posterior lobe and the vicinity of the ventricle seem to be the principal seat of abnormally located gray substance. Nevertheless, isolated patches have also been noticed in the anterior lobe—as by Meschede and Hoffmann. The former also found the same in the medullary centre of the cerebellum.

A case noticed by Klob stands by itself. He found a mass of *white* cerebral substance, the size of a bean, which hung from a pedicle between the two optic nerves, below the lamina perforata media.

Microscopic examination of the heterotopic gray substance shows, as a rule, formations otherwise similar to the elements of the normal cerebral cortex. The more or less numerous ganglion-cells, however, are pigmented or fatty.

The *clinical significance* of these anomalies is at present a matter of doubt; it is indeed true that they have been found exclusively in epileptics, idiots, or in persons otherwise mentally affected. But it must be taken into account that it is in post-mortems of just such persons that the brain is examined with especial care. All authors consider these malformations as congenital. Since many of these individuals have not showed any mental anomalies until later in life, some reserve is proper in interpreting these conditions.

But it is proper to mention that outgrowths of gray substance, especially in the ventricular region and in hydrocephalic subjects, have sometimes been found complicated with inflammatory processes (wounds, syphilis).

ATROPHY OF THE BRAIN.

Atrophy of the Corpus Callosum.

Reil, Arch. f. Phys. Bd. XI. 1812. S. 341.—*Chatto*, Lond. Med. gaz. I. 1845.—*Paget*, Med. chir. transact. Vol. 29. p. 55 et seq.—*Henry Mitchel*, Med. chir. transact. Vol. 31. 1848. p. 239.—*Foerg*, Die Bedeutung des Balkens im menschlichen Gehirn. München 1855.—Aerztliche Berichte der Wiener Irrenanstalt pro 1853. Wien 1858. S. 189.—*H. Langdon Down*, Med. chir. transact. 1844, 1861. p. 219–225.—*Poterin-Dumontel*, Gaz. méd. de Par. 1863. No. 2. p. 36–38. *H. Langdon Down*, Lancet 1866. V. II. No. 8 und Journ. of ment. sc. 1867. April.—*Birch-Hirschfeld*, Ueber einen Fall von Hirndefect in Folge eines Hydrops septi pellucidi. Arch. der Heilk. 1867. S. 481.—*J. Sander*, Ueber Balkenmangel im menschlichen Gehirn. Arch. f. Psychiatr. Bd. I. S. 128–142. 1868.—*Jolly*, Ein Fall von mangelhafter Entwickelung des Balkens im menschlichen Gehirn. Zeitschr. f. rat. Med. Bd. XXXIV. S. 4–14. 1869.—*Huppert*, Ein Fall von Balkenmangel bei einem epileptischen Idioten. Arch. d. Heilk. 1871. H. 3. S. 243.—*Malinverni*, Gazetta delle Cliniche 15, 1874 und Lond. Med. Rec. Nr. 73. 1874.

We consider in this chapter the entire or partial absence of the corpus callosum only in so far as it is the essentially abnormal condition present, and do not include secondary atrophy, caused, for example, by tumors, nor those cases in which large portions of the hemispheres are also wanting.

Etiology and Anatomical Changes.

Within the above mentioned limits, almost all the abnormal conditions now to be considered must be referred to retarded development during intra-uterine life, without our being able always to give a satisfactory explanation of their occurrence.

Towards the end of the fourth month of pregnancy, two lateral stumps normally appear on the surface of the hemispheres, which are the commencement of the corpus callosum. These grow toward one another and unite between the sixteenth and twentieth weeks.[1] This union probably takes place from before backward, so that the different forms of the anomaly, as described, can be explained in part by the period at which the arrest of development began.

1. Either no effort has been made to form the corpus callosum, and the entire system of transverse commissures is wanting, with the exception of the lamina cinerea, the optic commissure, and the anterior commissure.

Or, 2. The development of the commissure system was begun normally, but the communication between the two hemispheres does not extend backward for the normal distance,[2] but merely forms a rudimentary bridge of greater or less length and thickness. This rudiment may be either thinner or thicker than the normal genu corporis callosi with its surrounding tissue.

Or, 3. There may be a fissure in the corpus callosum which is otherwise normal in extent, no union having occurred at this fissure. Under these circumstances the separate portions of the structure may show different degrees of arrest of development, or the entire corpus callosum may be replaced by a thin, gauze-like or sieve-like plate of tissue (Huppert).

Most of the brains presenting this condition have also had dilated ventricles, so that the malformation has by many been referred simply to intra-uterine hydrocephalus. Nevertheless, the problem is not so simple. If the corpus callosum is entirely wanting, or if its rudiments contain no nervous structures (a fact which would have to be proved), then the radiating fibres of the corpus callosum will also be wanting, and accordingly the ventricles, whose roof is gone, become abnormally large, especially in their posterior cornua. In some cases, besides dilatation of the ventricles, other changes peculiar to hydrocephalus are found, such as thickening and granulation of the ependyma and disease of the choroid plexus. Yet these changes only point generally to an inflammatory affection within the cranial cavity, which may very well coexist with other etiological conditions without its *necessarily* following that one of the changes should be considered as the consequence of the other. In all this there is nothing incompatible with the supposition advanced by J. Sander in his very thorough work, that the defect in development is dependent upon original anomalies in the vessels.

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[1] *Reichert*, Der Bau des menschlichen Gehirns. Leipzig, 1859–61.

[2] The posterior end of the corpus callosum normally lies on a plane with the posterior end of the posterior central convolution.

After finishing this work I saw for the first time the abstract of Malinverni's remarkable case in the London Medical Record. The corpus callosum was found to be entirely wanting in a man who was, it was said, mentally sound and who died of an intercurrent disease. The septum lucidum was likewise wanting, and probably also the anterior commissure. The fornix itself was present. The inferior cornu was narrow, and the relation of the gray to the white substance, as also the weight of the brain, was said to be normal. (?) There is no distinct statement in regard to the posterior cornu. When it is stated that the gyrus fornicatus was also wanting, it is doubtless a mistaken way of expressing it. Its place was occupied by convolutions only differing from it in their shape or arrangement, but which might naturally include the same or perhaps even more elements. Unfortunately, I was not able to examine the original.

So far as the report permits us to draw a conclusion, this case seems to furnish decided evidence in favor of the positive influence of original anomalies of the vessels, and against the *necessity* of intra-uterine hydrocephalus. In Huppert's case, on the contrary, the atrophy was probably caused by a hydrocephalus, which occurred just as the groundwork of the corpus callosum was essentially completed.

Symptomatology.

The most varied functions have, at various times and without the least foundation, been ascribed to the corpus callosum. It is astounding that Lapeyronie, for example, on the basis of very bad observations, was able to gain partisans like Louis, Chopart, Saucerotte, and others, for his hypothesis that the corpus callosum was the seat of the soul.[1] Probably the corpus callosum serves physiologically to render possible a consistent and harmonious action of the mental functions of both hemispheres, since, as the principal transverse commissure, it anatomically unites their material substance. In all vertebrate animals, from birds downward, it is indeed wanting. Nevertheless, as Sander very correctly remarks, this can prove nothing against the supposition, since the defective mental development of those animals may well allow the absence of this or that organ which is necessary for higher animals.

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[1] *Longet*, Anat. et physiol. du système nerveux. Paris. 1842. T. I. p. 532 et seq.

From this point of view, it seems to me, certain of the postmortem changes agree tolerably well with symptoms present during life. A considerable number of these persons were either idiotic to the last degree, or at least slow-witted; at all events, the most had some mental defect which, it is true, could, in part at least, be referred to other concomitant cerebral diseases.

There are three cases, those of Paget, Jolly, and Malinverni, in which the defect of the corpus callosum was a purely accidental discovery in persons mentally sound. If the description given by Jolly, and his representation, are taken as guides, this condition seems to be explained by the fact that the genu corporis callosi was much thickened, while the splenium corporis callosi was indeed wanting, but that communication between the posterior lobes seems to have been possible through the forked posterior division of the corpus callosum, which was lost in its medullary substance, as is normally the case. Should another such case occur, the question would have to be decided in how far such thickened parts or the otherwise undisturbed portions of the commissural system had supplied the communication which could not follow the usual channel.

Coarser motor or sensitive defects need not in any case accompany atrophy of the corpus callosum. The mental defects sometimes present have of course nothing characteristic, so that the diagnosis cannot be made before the autopsy.

Atrophy of the Cerebellum.

Combette, Revue médic. T. II. p. 57. 1831.—*Cruveilhier*, Anat. pathol. Livr. XV. pl. V. Quoted from *Longet*, Anat. et phys. etc. T. I. p. 764 ff.—*Fiedler* und *Bergmann*, Ein Fall von Verkümmerung des Cerebellum. Zeitschrift f. rat. Med. 1861. S. 250–265.—*Duguet* und *Vulpian*, Deux cas d'atrophie du cervelet. Gaz. hebdom. 1862. No. 46.—*Moreau de Tours*, Deux cas de sclérose (Atrophie avec induration) du cervelet. Gaz. des hôp. 1863. I.—*Lallement*, Ein Fall von Atrophie des Kleinhirns. Schmidt's Jahrb. 1863. VIII. (Bull. de la soc. anat.

XXXVII. p. 190. Mars, Avril, 1862.)—*Meynert*, Ein Fall von Schrumpfung der Varolsbrücke und des Kleinhirns. Med. Jahrb. d. Ges. der Aerzte 1864. H. 4. S. 102–111.—*Pierret*, Note sur un cas d'atrophie periphérique du cervelet avec lésion concomitante des olives bulbaires. Arch. de phys. T. IV. 1871–72. p. 765 –770.—*Edw. Clapton*, Atrophy of the cerebellum. Transactions of the pathol. Soc. 1871. XXII. p. 20.—*Otto*, Ein Fall von Verkümmerung des Kleinhirns. Arch. für Psych. u. Nervenkr. Bd. IV. S. 730–746. 1874.—*Franz Fischer*, Eine interessante Hemmungsbildung des kleinen Gehirns. Ibidem Bd. V. H. II. S. 544–548.

While diseases of the cerebellum are rather common, it is rare to find a case of *extreme*, *uncomplicated*, simple, or sclerotic atrophy of that organ. The above bibliography contains all on this subject which I have been able to find.

Though these cases are few, they seem to afford important aid in forming an opinion in regard to the function of this organ.

Although not a few other cerebellar lesions cause symptoms similar to those of atrophy, and although the experiments of many physiologists are in harmony therewith, yet it might with more or less reason be urged, as an objection to admitting the material thus furnished, that the other organs in the posterior part of the skull were injured, and especially that sudden disturbances of circulation, tearing and pressure of these organs could not be excluded. This objection cannot be brought against the observations here adduced, so that they, in common with a careful selection of cases of softening and cystoid degeneration, form a valuable collection from which to judge of the physiological importance of the organ.

Slight degrees of atrophy are often noticed, both alone and as complications of other cerebral diseases, or as consequences of centres of disease (foci) in the organ itself. But in the present state of knowledge these are of no special interest to us. In all these cases, especially in uncomplicated atrophy, there may be no definite symptoms. But if there are any, they either cannot be localized, or it is customary to consider them dependent upon the primary lesion as effects of the focus of disease, and not upon the atrophy.

Etiology.

In the cases reported by Lallement and Otto the atrophy was certainly *congenital*, while in Combette's case (Alexandrine Labrosse) this was probably only partially true. It is deserving of remark that, among the few cases on record, fright is twice (Meynert and Pierret) definitely assigned as the cause. In Clapton's case the nervous symptoms appeared after measles and gradually diminished in severity. The other authors have very little to say about the etiology.

Pathology.

Anatomical Changes.

In Combette's celebrated case, according to a literal translation given in Longet's work, the following conditions were found:

> On cutting through the tentorium, there flowed out a large amount of serum, which had filled the posterior fossa of the skull. In place of the cerebellum, there was found a hemispherical gelatinous membrane, which was attached to the medulla oblongata by two membranous and gelatinous pedicles. On these pedicles I found two small masses of isolated and separate white substance, of the size of peas. The corpora quadrigemina were intact. There was no fourth ventricle; there was no trace of a pons, nor was there any cavity corresponding to the situation which it should have occupied. All the cerebral arteries were present, and of their normal size.

This case is unique so far as relates to the total disappearance of the organ. Even in other cases, however, the cerebellum seems to have lost one-half or more of its weight. Lallement mentions a case in which the left cerebellar lobe, with its middle and superior peduncles, was reduced to the size of a nut, and the transverse fibres of the pons were atrophied, as well as the right corpus striatum and the right olivary body.

In Duguet's case the cerebellum weighed ninety-five grammes (℥ iii.), about half the normal weight. The atrophy, so far as relates to the nervous elements, was bilateral and general, since the portions which seemed to be of normal size and structure showed under the microscope a very well-marked sclerosis. Very like this is the report made by Clapton. In the Fiedler-Bergmann case, too, the organ was reduced to at least one-half its volume, and, though rather more nervous elements were found in it, yet there was a very great degree of sclerosis, which could not have been consistent with much activity in this part of the encephalon. No statement is made in regard to the condition of the pons.

Meynert, however, accurately describes a similar degeneration in the *pons* (which was also noticed in several of our cases), and at the same time asserts that it must be considered as a *secondary degeneration*, and not as the starting-point of the affection. The cerebellum itself was much altered, especially in all parts of the right side. The posterior pyramids were also implicated, as well as the pons and the crus cerebelli ad pontem. Microscopic examination showed that in the cerebellum the medullary substance was especially affected, while the gray substance was less altered. Finally, in Pierret's case there was likewise an intense degree of sclerotic atrophy, which affected chiefly the vertical diameter of the organ. The microscope, however, showed that all parts were affected, excepting the corpus rhomboideum, while the gray substance was the one principally involved. The transverse fibres of the pons and both olivary bodies were also atrophied and replaced by connective tissue. In regard to the latter, it is worth mentioning that they preserved their external form.

Otto found that, in his case, the hardened cerebellum, including the pons and the medulla oblongata, weighed only twenty grammes (℥ v.). The left lobe was the one most atrophied, and, corresponding thereto, the pons on the left side was narrower than on the right.

It is worthy of notice in this case that there were no complications recognizable externally, but that the space occupied by the organ was proportionately reduced by a general hyperostosis of the occipital bone. In view of the course of the disease, there can be no question that it was a congenital defect. It is all the more to be regretted on this account, that Otto made no report of the microscopic conditions present. In view of the peculiar combination of symptoms which the patient showed, it was of importance to know whether the rudiment of the cerebellum consisted essentially of normal tissue or not.

Symptomatology.

If a remarkable uniformity is found in the account of the anatomical changes, this is even more noticeable on comparison of the symptoms reported. Singularly enough, this very evident fact has often been distorted into appearing just the opposite, the original statements of the authors being generally taken at second or third hand, and also being viewed through colored glass, by the writers who thus misrepresent them.

Combette's case has thus experienced the most remarkable fate. By one writer the twelve-year-old girl under consideration is represented as having been quite healthy and active; by another she is declared to have suffered from slight motor disturbances, not at all proportionate to the cerebral defect. Some authors, influenced by a theory mentioned below, ascribe special importance to the fact that the child masturbated excessively. Nevertheless, the original report in regard to this case of complete loss of the cerebellum is very different from any of these, and, as we find its principal phenomena reproduced more or less entirely in nearly all our cases, we feel justified in giving it prominence.

The little Labrosse girl was extremely *weak-minded*, and suffered from *epileptic attacks.* Furthermore, she did not learn to stand and walk until she was five years old. At seven years of age she had "*beaucoup de faiblesse dans les extrémités*," and often fell down. Toward the end of her life—at the age of twelve years—she was bedridden for three months, and could scarcely move her legs. Finally, there was disturbance of speech ("*l'articulation nette des sons était impossible*").

We find here then as *characteristic symptoms*, first, a great degree of *motor disturbance*, which affected also the articulation; then serious *mental deficiency*, to which were added spasms and onanism.

Now, there was likewise a high degree of motor disturbance in the cases reported by Meynert, Pierret, Fiedler, Clapton, Duguet, and Moreau. In no case was the disturbance designated as paralysis—only Pierret speaks of weakness of one upper extremity—but the description almost always recalls the analogous symptoms of tabes dorsalis. Most authors expressly call the symptom *ataxia* or *disturbance of co-ordination;* others state that the patients could indeed walk, but only slowly and carefully; that they fell frequently, especially backward; that in walking they seized hold of objects within reach. All these patients had also persistent, or at least temporary (Fiedler), *disturbances of speech.*

No motor disturbance was noticed by Lallement and Otto. Otto even states that his patient was especially adroit and active, yet he was somewhat "impulsive" in his motions—a fact which had also attracted the notice of former observers. Whether this peculiarity was entirely of a psychical nature, or the expression of a disturbance of co-ordination, cannot of course now be determined.

Weakness of mind, even extreme idiocy, characterized the patients of Clapton, Otto, and Fiedler, while Pierret's patient suffered from weakness of memory. Here and there the statements, especially in regard to the mental condition, are imperfect, or are derived from incompetent attendants.

Epileptiform convulsions occur as irregular accompaniments of so many cerebral and non-cerebral affections that their occurrence in a number of our patients seems very natural, without, however, having any special significance.

Among the symptoms only occasionally noticed, and hence having as yet rather an accessory character, we may mention: extreme *analgesia* (Fiedler) and slight *disturbance of sensation* (Pierret). But from the intimate relations of the cerebellum to the posterior columns of the spinal cord it might be supposed *à priori* that more careful examinations would perhaps in other cases also have shown deficiency of sensibility.

Finally, the onanism of the little Labrosse deserves special mention. We turn now to the significance of the symptoms.

It is well known what a sensation Gall produced, and how much attention he excited in literature, through an attempt to prove that the cerebellum is the seat of the sexual instinct of physical love. We cannot here follow the phases of the contest over this proposition, and refer the reader to the works of Longet,[1] Leuret,[2] Huschke,[3] and Lange.[4] At all events this much is certain, that the pretended proofs of Gall were so extremely insignificant that it may well be wondered why such an immense amount of work by the best men could be and had to be expended in order to refute them.

We do not wish to discuss the question whether the so-called sexual instinct is a quality separable from the other cerebral functions, and in so far capable of localization. But the fact that this little girl masturbated has led to the conclusion, both formerly (Longet, for instance) and more recently, that her sexual instincts were developed to a high degree, and this very argument has been brought forward in opposition to the theory of Gall. This conclusion, however, cannot be said to rest on a basis of facts, unless we admit that improper, or even constant handling of the genitals, of itself, in all cases, justifies the conclusion that sexual ideas, or at least sensations are present. At the same time such a supposition is rendered very doubtful by the persistent friction practised by idiots, who most probably neither have any ideas connected with it, nor any sensory excitation worth mentioning resulting therefrom. But onanism, if we are justified in calling it such, is very often met with during the earliest years of life, especially in little girls, at a period when there can be no question about the existence of a sexual instinct. I have always considered this manifestation to be the mediate or immediate effect of a sensory irritation, and compare it, for example, to the practice of incessantly picking one's nose, which is popularly regarded as a sign of intestinal worms, but is sometimes practised incessantly where there are no worms.

The question arises, in how far the most essential and constant symptom of atrophy, namely a *defect in the co-ordination*

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[1] Anatomie et Physiol. etc. T. I. p. 732–769.

[2] *Leuret* et *Gratiolet*, Anatom. comp. du syst. nerv. T. I. p. 425 et seq. and 546 et seq.

[3] Schädel, Hirn. und Seele. p. 65 et seq.

[4] Geschichte des Materialismus. Leipzig und Iserlohn, 1873–75.

of motion, is in harmony with the results of experimental physiology. For, influenced by reasons mentioned at the beginning of this paper, and in view of the ground covered by other divisions of this work, we shall not here attempt to collect and estimate the value of other pathological experiences.

Here it appears that the results of those observers who have made the most extended experiments fully agree in one respect —all found Flourens'[1] statement confirmed—that artificial injury of the cerebellum disturbed the harmony of the movements.

I may mention here Longet, Leven, Gratiolet and Ollivier,[2] Lussana,[3] Goltz,[4] and to these investigations I may add my own.[5] One may notice that this is the very point in which all *clinical* observations agree, excepting two. Clinical experiences as well as physiology agree in teaching that serious interference with the function of the cerebellum does not indeed destroy motion, but is shown by a peculiar disorder therein.

In regard to individual symptoms, it is true, the above observers do not agree, just as their results also differ from those of other authors. This depends in part upon the methods employed, in part upon the animals experimented on. Since the other phenomena which they called forth—namely, amaurosis, strabismus, and acute disturbances of equilibrium (rotatory motions)—are wanting in atrophy of the organ, we need not occupy ourselves with them. It is more important for us to consider what significance is to be attached to the two cases (Lallement and Otto) in which there were no motor disturbances. It is a custom, as common as it is unscientific, on the evidence of such exceptional cases, to deny that certain cerebral organs possess functions which, according to all other experiences, belong to them. The reason for this undoubtedly lies in the fact that, usually, authors study only one or another organ of the brain;

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[1] Comptes rend. 1860.

[2] Arch. gén. 1862. Nov., Dec.

[3] *Lussana e Lemoigne*, Fisiol. dei centri nervosi, etc. Pad. 1871. Vol. II. p. 179 et seq.

[4] Beiträge zur Lehre von den Functionen der Nervencentren des Frosches. Berlin 1869.

[5] Untersuchungen über das Gehirn. Abhandl. IX., X, XI.

if this method of reasoning were followed out, it would inevitably lead to the conclusion, founded upon more or less numerous and important exceptional cases, that the entire brain, including the medulla oblongata, is a superfluous organ, a mere article of luxury, which we may have or not—somewhat like a luxurious growth of hair. Lallement's case is especially deserving of mention in this connection. In it there was also atrophy of the opposite corpus striatum and of the olivary body without motor disturbance, and no doubt was raised in regard to the motory function of these organs. In truth, later investigations (Flechsig) prove that within physiological limits there may be great individual variations in the course of the central paths of conduction with reference to the two halves of the body, and from this, but yet more from my own investigations, may be deduced the possibility of similar relations in the combinations of these conducting paths. Really, the defect in Lallement's case was without doubt congenital and marked by (vicarious) hypertrophy of the other hemisphere of the cerebellum and cerebrum. The lesion in Otto's case is likewise to be looked upon as congenital; but this case is not worthy of our further consideration, for reasons mentioned above.

As to the condition of mental weakness, which has been several times referred to, it is sufficient to point to the conviction which is continually gaining ground, that all great injuries and defects of the brain, irrespective of locality, according to their peculiar circumstances sooner or later usually produce evident changes in the mental functions.

Complications and Diagnosis.

As frequent complications of atrophy of the cerebellum we may mention atrophy of the cerebrum, of the transverse fibres of the pons, and of the olivary bodies. By some authors a causative connection, in the nature of secondary degeneration, is considered certainly to exist between these various anomalies, but the proofs adduced in support thereof are not always equally convincing. The view which seems best grounded is that according to which the transverse fibres of the pons, passing through the middle crus cerebelli, are considered to be commissures of the cerebellar hemispheres, and their atrophy is thus logically explained.

Simultaneous atrophy of one cerebral and one cerebellar hemisphere usually occurs where such a combination is found on opposite sides. Luys and Meynert believe that corresponding to this they have found the anatomical proof that large bundles of fibres from the cerebral peduncle in part pass with a simple decussation through the superior peduncle, in part pursue a peculiar looped course through the pons and the middle cerebellar peduncle to the opposite hemisphere of the cerebellum. These questions are at present the subject of anatomical and experimental discussion, so that a further consideration of them is not advisable. We only remark that unilateral atrophy of the cerebellum, in case of unilateral atrophy of the cerebrum, is more frequently wanting than present, and that in some cases it occurs on the same side, not on the opposite one. For further particulars we refer the reader to the statements of Meynert,[1] Huguenin,[2] and Schroeder van der Kolk.[3]

The *diagnosis* of atrophy of the cerebellum is always attended with great difficulty. The rarity of the affection is to be remembered, and should make one slow in deciding the diagnosis. An atrophy must be difficult to distinguish from *other chronic affections* of this organ. Yet headache and vomiting—so common in diseases which diminish the capacity of the posterior fossa of the skull—seem to be rather rare in simple atrophy.

It is quite possible to mistake this disease for *tabes dorsalis*, since both present the symptoms of disturbance of co-ordination. Yet assistance may be obtained by careful consideration of the course of the malady, especially those symptoms relating to the bladder and the lancinating pain, which are rarely absent in advanced cases of tabes. A fact not heretofore published, which has been firmly established by me, may also be made use of, namely, that one of the earlier though certainly not absolutely constant signs of tabes, is the appearance of a zone anæsthetic to a light touch, on a level with the third, fourth, fifth, or sixth dorsal vertebra.

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[1] Skizze des menschl. Grosshirnstammes, etc. Arch. f. Psych. Bd. IV. p. 387–431.

[2] Allgem. Pathol. der Krankh. d. Nervensyst. Zürich 1873. p. 182–185.

[3] Waarneming van eene Atrophie, etc. Verhandel. der Eerste Klasse van het Nederl. Institut III. Reeks v. Deel. p. 31, 1853. (A very full abstract by Virchow in Jahresbericht for 1853.) See also *Virchow*, Schädelgrund.

Among the other affections here claiming our attention are multiple cerebro-spinal sclerosis and bulbar paralysis. Both diseases may give rise to error from the peculiar disturbance of speech attending them, the first also by the symptoms of motor disturbance.

In fact, a correct diagnosis between *multiple sclerosis* and our disease at an early stage is scarcely possible, because sclerosis may also affect the cerebellum or parts which most probably contain its commissural and connecting fibres. Disturbances of co-ordination, epileptic attacks, and mental symptoms, in addition to disturbance of speech, are regularly, or at least often, observed as symptoms in sclerosis in patches. Nevertheless, upon longer observation the diagnosis can be made, for the course of development and the picture of sclerosis are sufficiently characteristic, notwithstanding the great variety of symptoms. At first sclerosis shows an evident, and later a very great diminution of general muscular power; then trembling of the limbs at some stage of the disease never fails, contractions are found, and finally peculiar tetanic attacks supervene—all symptoms not seen in pure atrophy of the cerebellum.

Even on superficial examination our disease could not well be mistaken for *bulbar paralysis*. In bulbar paralysis the localization of the marked paralytic symptoms in the lips, tongue, and œsophagus is so peculiar as to prevent such a mistake. Besides, trouble of co-ordination is not found, while a complication with the symptoms of progressive muscular atrophy is not rare.

General Paralysis of the Insane.

Bayle, Recherch. sur les malad. mental. Paris, 1822.—*The same*, Maladies du cerveau. Paris, 1826.—*Delaye*, Considérations sur une espèce de la paralysie, etc. Paris, 1824.—*Calmeil*, De la paralysie considérée, etc. Paris, 1826.—*Falret*, Recherch. sur la folie paralytique. Paris, 1853.—*The same*, Arch. générale. 1858.—

Duchek, Ueber Blödsinn und Paralyse. Prager Vierteljahrsch. XXIX. Band 1851. I. Band S. 1.—*Baillarger*, Ann. méd. psych. 1852–1862.—*Joffe*, Ueber Geisteskr. u. Paral., Zeitschr. d. K. Gesellsch. der Aerzte zu Wien. XIII. Band S. 675. 1857.—*Wedl*, Beitr. zur Path. der Blutgef. Wien. Sitzungsber. XXVII. 265. 1859.—*L. Meyer*, Die allgemeine progressive Gehirnlähmung. Eine chronische Meningitis. Berlin, 1858.—*The same*, Ueber die Bedeutung der Fettkörnchen, etc. Arch. für Psych. und Nervenkrankh. III. Band S. 1–65 und S. 242–311. 1870.—*The same*. Die patholog. Anat. der Dement. paral. Virch. Arch. LVIII. Band 1873.—*Erlenmeyer*, Die Gehirnatrophie der Erwachsenen.—*Rokitansky*, Lehrb. der path. Anat. II. Band. Wien, 1856. S. 463 ff.—*Austin*, A pract. acc. of general paral. Lond., 1859.—*Parchappe*, De la fol. paralyt. Paris, 1859.—*Westphal*, Ueber Erkrankung des Rückenmarks bei der allgem. progr. Paralyse der Irren. Virchow's Arch. XXXIX. und XL.—*The same*, Ueber den gegenwärtigen Standpunkt der Kenntnisse von der allgemeinen progressiven Paralyse der Irren. Arch. f. Psych. und Nervenkrankh. I. Band S. 44–95, 1868.—*The same*; Einige Beobachtungen über die epileptiformen und apoplektiformen Anfälle der paralytischen Geisteskranken mit Rücksicht auf die Körperwärme. *Ibidem*, 337–386.—*Th. Simon*, Ueber den Zustand des Rückenmarks in der Dementia paralytica, etc. Arch. f. Psych. und Nervenkrankh. I. Band S. 583–625 und 328–363. II. Band S. 109–152. 1870.—*The same*, Die Gehirnerweichung der Irren. Hamburg, 1871.—*v. Rabenau*, Ueber die Myelitis der Hinterstränge by Geisteskranken. Arch. f. Psych. und Nervenkrankh. III. Band S. 697–710. 1872.—*The same*, Ueber das Verhalten der Körnchenzellen-Myelitis, etc. Ibidem IV. Band S. 317–334. 1874.—*Th. Meynert*, Ueber die Hirnrinde und die Rarefaction ihrer Nervenkörper bei Geisteskranken. Wiener med. Zeitung, 1866, 22, 28.—*The same*, Studien über das pathologisch-anatomische Material der Wiener Irrenanstalt. Vierteljahrsch. für Psych. 1868. Heft 3 und 4.—*The same*, Das Gesammtgewicht und die Theilgewichte des Gehirns, etc. Vierteljahrsch. f. Psych. 1867. Heft 2.—*Lubimoff*, Studien über die Veränderungen des geweblichen Gehirnbaues, etc. Virchow's Arch. LVII. Band 1873.—*The same*, Beiträge zur pathologischen Anatomie, etc. Arch. f. Psych. IV. Band. S. 579–600. 1874.—*Meschede*, Ueber die der paralytischen Geisteskrankheit zu Grunde liegenden pathologisch-anatomischen Veränderungen. Allgem. Zeitschr. f. Psych. XXIX. Band. S. 587. 1873. Virch. Arch. Bd. XXXIV. u. LVI. u. a. and. Ort.—*Huguenin*, Zur pathol. Anat. der Dementia paralytica. Correspondenzblatt für Schweiz. Aerzte. 1873. Nr. 21, 22, 24 und Verhandl. d. schweiz. naturforsch. Gesellsch. 1872–73. S. 272.—*Magnan* et *Mierzejewsky*, Des lésions des parois ventriculaires et des parties sousjacentes dans la paralysie générale. Arch. de physiol. norm. et path. 1873. I.—*v. Krafft-Ebing*, Ueber die klin. different. Diagnose zwischen der durch Periencephalomeningitis bedingten, etc. Allgem. Zeitschr. für Psych. XXIII. Band S. 181. 1866.—*Schuele*, Sectionsergebnisse bei Geisteskr. Leipzig, 1874.—*The same*, Beiträge zur Kenntniss der Paralyse. Allgem. Zeitschr. für Psych. XXXII. Band. S. 581. 1875.—*Mierzejewsky*, Etudes sur les lés. cérebr., etc.

Arch. de physiol. Sér. II. T. II., p. 195. 1875.—*Marcé*, Etudes clin. et anat. path. sur le dém. etc. Gaz. méd. 1863.

By the name general progressive paralysis of the insane, softening of the brain, paralysie générale incomplète, progressive, folie paralytique, or dementia paralytica, is understood a diffused disease of the brain, and often also of the spinal cord, which is characterized by a peculiar combination of psychical changes with motor disturbances in the muscles of different parts of the body, which has a chronic course and ends in death. Inasmuch as marked atrophic conditions of the cerebellum are very frequently present, and, indeed, always exist in protracted cases, this disease belongs to our division. On account of the special importance which the affection possesses in its earliest stage not only for the physician and the afflicted person, but also for society at large, we will describe it somewhat at length. Nevertheless, in view of the limited space at our disposal, we must plainly state that the following description is not intended for alienists by profession, and that it makes no pretensions to being considered a complete monograph, especially in view of the extraordinary extent to which the literature of progressive paralysis has grown within the past few years.[1]

Etiology.

The etiology of progressive paralysis agrees in many respects with that of other forms of insanity, yet differs from it in many interesting particulars. It has been and is still declared by many that there is either no evidence of *heredity* in general paralysis, or, at least, that it plays an unimportant rôle. But the same standard is not employed by all authors in judging of an inherited predisposition; the proper weight has not been given in particular to the frequency with which a transformation of nervous disease occurs, and to the influence of alcoholism in the ancestors. Thus Grainger Stewart, in opposition to the first view, states the per cent. of cases wherein heredity can be traced as forty-seven and a half, which is, however, much too large. Greater unanimity prevails in regard to the relative predisposition of the two sexes. It is generally acknowledged that by far the greater proportion of cases is found in males, though, indeed, as in all statistics, the numbers differ considerably with different observers. According to the statement of W. Sander[1] there should be one woman to 7.5 men. This extraordinary proportion has not only a relative but also an absolute importance, since the paralytics include one-quarter of all the insane among males. In close connection with this circumstance is the fact that those of more mature age are more frequently thus affected. Under the age of twenty years paralysis scarcely occurs; it is most frequent between thirty and forty-five. Some authors Conolly, Reye) have placed the greater number in the sixth decade.

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[1] Bibliography to the year 1867, by v. Krafft-Ebing, zur Geschichte und Literatur der Dementia paralytica. Allgem Zeitsch. f. Psych. Bd 23. S. 626–646.

We can hardly go amiss in referring these facts, which find their analogue in the conditions prevailing in tabes dorsalis, to the influence of the mode of living. The male sex is not only exposed to greater bodily and mental exertions and injuries, but it endeavors to console itself for these by more liberal indulgence in pleasures which make quite a demand upon the nervous system. Probably the combination of excessive labor with excesses *in Baccho et Venere* is the most common cause. The influence of sexual excesses can be recognized in females also.

It must be remembered that especially in paralysis all figures have only a relative, that is, a purely local value. It is a well-known fact that in large cities the disease is much more common, and this is especially true in regard to its prevalence among females. Every asylum must hence give different statistics according to the territory from which it is filled, quite independently of the influence of diagnostic accuracy and bias.

Besides this, *injuries of the head*,[2] *constitutional syphilis, and the influence of acute febrile diseases*, give rise to conditions of the nervous system which either indirectly, through the membranes, or directly through alterations of the nervous substance, as yet unknown, favor the development of the disease.

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[1] Die paralytische Geistesstoerung beim weiblichen Geschlecht. Berliner klin. Wochenschrift. 1870. 7.

[2] *von Krafft-Ebing*, Ueber die durch Gehirnerschütterung und Kopfverletzung hervorgerufenen psychischen Erkrankungen. Erlangen, 1866.

Pathology.

Anatomical Changes.

For many years the study of the pathological anatomy of dementia paralytica has been the chief object towards which our ablest alienists have directed their labors. Although much that is valuable has been gained thereby, there has been but little agreement in the results reached, and, perhaps for that reason, in the views adopted concerning the nature of the affection. It is rather noticeable that even those authors who have abundant material at their command maintain quite different views. It would be wrong to undervalue, on that account, the work accomplished, for the facts obtained will, without doubt, in time be brought together into their appropriate positions. Nevertheless, in the present situation of things, one experiences great difficulty in presenting the case aright, since each author, as usual, is inclined to consider his own discoveries the most conclusive—a circumstance which seems also to have prevented the authors from understanding one another. Many investigators besides maintain, as we believe not unreasonably, "that the term *general paralysis* is a clinical collective name, just as puerperal fever once was" (Schuele).

At the very outset we must emphasize the opinion—which it is to be hoped is now generally accepted—that *all mental diseases* are nothing else than the outward functional manifestations of *cerebral diseases.* Although in some cases—whose number is constantly diminishing—no post-mortem changes are found within the cranium, yet this would not be a proof of the integrity of the organ, even if its microscopic examination were accompanied with less difficulty.

But, if we turn to *positive* data, the relative bulk of the brain, as shown to the eye and by weight, is the first thing to attract our attention, not only on account of its close relationship to our subject, but also on account of the greater objectivity of the method of examination, and on account of the agreement of authors. It may be considered as settled, and my own experience agrees perfectly with this view, that wherever the disease does not run a rapidly fatal course—that is, *in the protracted cases* with which we have first to occupy ourselves—there is always *atrophy of the organ*, an atrophy which can be recognized by simple inspection, frequently also by weight.

On opening the head the dura mater is seen to lie in folds over the frontal lobes, without, however, necessarily indicating atrophy of the anterior lobes, for the folds are found at this place also in atrophy of other parts, if the body, as is usual, lies on its back. The pia mater is either universally or locally œdematous, especially in the sulci; a great collection of water is often found over the parietal and occipital lobes; the temporal is almost always free; the frontal is frequently free, or less affected. I have not been able to fully convince myself that this condition depends entirely upon the dorsal position of the body, for it does not vary if the body has lain upon the stomach up to the moment of examination. When all the circumstances are taken into account, this œdema of the pia mater must be considered as a dropsy *ex vacuo* in consequence of the atrophy of the brain.

The *ventricles*, including the third ventricle, are enlarged—often greatly dilated, especially in their cornua. The *ependyma* is remarkably often more or less thickly covered with larger or smaller granulations. In some cases they are so thick that the surface looks like shagreen or a cat's tongue. Bayle called attention to these granulations. More lately they have been studied by Virchow (gesammelte Abhandlungen), Joire, Magnan, and Mierzejewsky. Joire thought the essence of the paralysis was to be found in the disease of the ventricles. Magnan and Mierzejewsky, on the other hand, showed the presence of the same process on the cortex and in the ventricles, pervading the centre of the organ from without inwards as well as from within outwards.

The great medullary mass seems relaxed; the surface of the section falls in or is easily pushed together. The surface of the large ganglia is rough, as if covered with finger indentations, and its surface paler, more withered than normal.

Atrophy of the convolutions is shown by the collections of water in the sulci; on more careful examination, individual gyri are seen, more or less diminished in size, their crests being often only as thick as the back of a knife. Some state that the frontal convolutions are especially affected, others are less exclusive. I must agree with the latter, and only remark that I have found the most frequent and comparatively the most advanced atrophy in the posterior central convolution. The atrophy sometimes affects the white and sometimes the gray substance of the gyri more severely; the layers of the latter often disappear.

The result of this loss is shown in a *diminution of weight*, which often sinks below 1,000 grammes (2 lbs. 8 oz.); though so great loss of weight is not the rule, nor is it specially frequent. We owe important observations on this point especially to Parchappe and Meynert, who have, however, by no means exhausted the subject.

As to the *weight of separate portions*, Meynert states that the cerebellum is unaffected, while the cerebral expansion is greatly, the base less, atrophied. But the frontal portion of the cerebrum, in which Meynert includes the anterior central convolution, is most affected.

The pia mater, besides the œdema already mentioned, shows the most varied kinds, degrees, and stages of pathological change. Most frequently there is found a diffused dullness, sometimes whitish, sometimes yellowish, and thickening in irregularly shaped patches by the side of the longitudinal fissure and in the vicinity of the larger vessels. Another time, more regularly rounded patches, from the size of a millet seed to that of a lentil, are found scattered over entire lobes; between them, especially where the membranes are reflected, spots of thickening, rarely the size of a bean, which on inspection might easily be mistaken for a collection of pus, but when divided are found to be rather firm. Not seldom the thickening is more general, and extends over the whole convexity, including the medial surface; or the envelope of individual lobes alone may be affected, the ones most frequently involved being the anterior and parietal lobes.

Wherever there is œdema the pia mater can be easily removed, usually without tearing the cortex; but immediately after this is done, an unusually large number of small holes will often be noticed in the latter, corresponding to vessels which have been pulled out. Others claim that they have seen the surface sprinkled over with warts and granulations (L. Meyer) (to be distinguished from the so-called epithelial granulations of the pia mater, Bayle and L. Meyer), representing the remains of a former inflammation.

On the other hand, if there is no œdema, the pia mater often cannot be removed from the diseased places without causing more or less loss of substance, so that the cortex has the appearance of having been gnawed by mice.

Magnan has sought to diminish the significance of these facts, and to show that they represent a normal condition, by the objection that after the injection of water into the cerebral vessels these adhesions give way. This objection, however, loses in weight very decidedly when we consider the predilection which these adhesions have for certain regions (frontal portion, crest of the convolutions). Furthermore, as was mentioned above, this firm adherence is also wanting in cases of non-artificial œdema of the pia mater.

The *dura mater* is also affected remarkably often, so much so that for a while every one was inclined to look upon its affection as the essential lesion. The membrane often adheres so firmly to the skull that the latter cannot be removed separately without using great force and crushing the brain. Therefore it is better to remove dura mater and bone together. The membrane may present all varieties of dullness, thickening, and deposits upon its surface, these being generally more diffuse than in the pia mater. Neoplasms of osseous tissue (osteomata) are sometimes, though rarely, encountered; suppurative changes are perhaps still more rare; on the other hand, effusions of blood, of greater or less extent, are far more frequent. Sometimes the entire dura mater of both hemispheres, not excepting their bases, is covered with innumerable larger or smaller flattened extravasations, which have all shades of color between yellow, red and black. More rarely there are large masses of blood, or sacs of such size as by their pressure to produce hollows in the convolutions.

According to Heschl and Virchow, the effusion takes place, as a rule, between the lamellæ of the primarily inflamed and thickened membrane; in which case a rupture inward is all the less excluded, as the pachymeningitic lamella, which is generally visceral, is formed of tissue as thin as a cobweb. Huguenin, on the contrary, supports the view that the hemorrhage is primary, and comes from the intra-pachymeningeal veins, which have undergone fatty degeneration.

A portion of the larger hemorrhages must, without doubt, be looked upon as traumatic hæmatomata. Paralytics are especially liable to injuries of the head, and the vessels of their cerebral membranes are particularly inclined to rupture.

Though these affections of the dura mater are so frequent, still they cannot be looked upon as constituting the specific lesions of general paralysis. There are paralytics enough who have a perfectly tender dura mater, and other post-mortems again show pachymeningeal changes, especially hemorrhages, without the peculiar symptoms of general paralysis having appeared during life. The large hæmatomas of paralytics are not generally revealed by the symptoms of cerebral pressure otherwise belonging to them, since the œdema which exists will be absorbed in proportion as the blood is poured out (Huguenin).

The *skull*, apart from occasional congenital anomalies and the remains of old injuries, presents very uncertain pathological changes. The conditions most frequently found are general thickening of one or more bones, especially of the frontal bone; also all sorts of exostoses and growths; finally, sclerotic degeneration; but more frequently the result is negative, or of little importance.

Like all other changes in general paralysis, the condition of the blood-vessels is very variable. Most frequently in these old cases, on section of the medullary substance, a large number of abnormally dilated red vascular cavities is seen—the *état criblé* of the French. These often correspond to the sections of miliary aneurisms. Otherwise the substance seems rather pale.

The larger vessels of the pia mater, likewise, are rarely very full. On the other hand, it is common to find patches of redness on the surface, caused by injection of the smaller vessels and capillaries, in the midst of very pale surroundings.

The changes found in cases which have run a rapidly fatal course are very different as described by Calmeil,[1] and more recently by L. Meyer.[2] Here the dura mater tightly surrounds a brain much swollen, especially in its anterior part. The convolutions are diminished; the sulci are narrowed; the surface of the gyri is colored a bright red, often rather spotted and grained; the thickness of the cortex is increased. On section, strong injection is seen, and there are many small centres of softening.

Microscopic examination of the brain shows less sharply defined differences between recent and old cases.

The most marked changes and those pretty generally recognized by the most recent observers are found in the *capillaries*, the *intermediate vessels*, and their immediate neighborhood. First the nuclei of the vascular wall seem increased in number; then masses of cells are seen adhering to them, more or less filling the perivascular space, and in places dilating it. These cells also wander out into the parenchyma. They resemble and probably are nothing else than colorless blood-corpuscles. Here and there single red blood-corpuscles are seen among them, and then regular small extravasations, in all stages of pigmentary change, appear, filling the lymph-spaces for a distance. The vessels themselves are filled and crowded with blood-corpuscles.

Gradually the walls of the vessels are said to pass into a fatty, in some cases an amyloid or colloid degeneration (the cells participating), such a change, however, not being so regularly found as that previously mentioned. It is still a very unsettled question whether there is a new formation of vessels.

According to Mevnert, Lubimoff, and others, there are developed in the *parenchyma* numerous "spider-cells," connected with the vessels by their processes, which themselves again fall into degeneration, and whose pretended rôle we will mention farther on. Others are, on the other hand, rather inclined to look upon the greater portion of these spider-cells as coagulations, artificial products, etc. The main substance of the parenchyma, after longer duration of the disease, is found in a more striated than granular condition, not rarely filled with small cavities (vacuoles)

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[1] Loc. cit., p. 381 et seq.

[2] Die pathologische Anatomie der Dementia paralytica. Loc cit.

Mierzejewsky lately speaks of peculiar structures in this substance, which he considers to be hypertrophied axis-cylinders. I have myself produced such things *artificially*, by cerebral injuries, and demonstrated them a year ago in Geneva. In the vicinity of the diseased focus appear numerous bodies of considerable size, with sharp outlines, appearing round, or sometimes rather conical, on section, capable of being strongly tinted by carmine, and without a nucleus. Sometimes they are grouped closely in the parenchyma, sometimes are scattered in the vacuoles, and sometimes the vacuoles are empty. I also suggested the probability that these were hypertrophied axis-cylinders; yet I am disposed to speak with less certainty, as I have not sufficient proof thereof from preparations that have been "teased apart."

The *ganglion-cells*, finally, undergo various forms of degeneration. So far as I can judge in regard to the still rather uncertain facts, fatty degeneration, or sometimes rather a pigmentary degeneration of a very large number of the cells, is a condition almost always encountered. More rarely a swollen and sclerotic condition of the nerve-cell is found.[1]

Meynert and Lubimoff suppose the order of events to be as follows: the perivascular lymph-spaces are compressed by hyperæmia of the smaller vessels, and so the lymph, which should flow off, is retained in the network of lymph-cells (spider-cells, Zeiter's cells), and imprisoned in the parenchyma, where it works further injury. Lubimoff believes he can go farther, and make the sympathetic nerve responsible for the hyperæmia.

Westphal,[2] finally, disputes the value of all the conditions described, inasmuch as they may be wanting in paralysis, and may be present in other diseases of the brain, or even in the brains of healthy subjects. In opposition to him we would merely declare it as our opinion that the amount, in bulk, of the changes present and the extent of the destruction of substance caused thereby, are the only criteria of their significance. But it may be conceded, on the other hand, that no *specific* change characteristic of general paralysis has yet been proven, and that the clear criticism of Westphal has certainly rid the subject to a considerable degree of unauthorized elements. Without pretending in the least that these questions are settled, we may thus formulate the view which at present seems the most probable, viz., *that general paralysis, in the more restricted sense, and so far as concerns the brain, is a chronic, or sometimes rather a sub-acute interstitial* (*peri*) *encephalitis, which in course of time leads to destruction of the ganglion-cells and to atrophy of the brain.*

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[1] Mr. *Weller*, second physician of the asylum conducted by me, will soon publish a detailed work in regard to the discoveries here made. Complete bibliography by *Mierzejewsky*, loc. cit.

[2] Ueber den gegenwärtigen Standpunkt, etc., loc. cit.

The *membranes of the spinal cord* undergo, though more rarely, changes similar to those of the brain.

The condition of the *spinal cord* itself has been the object of many and careful examinations since the labors of Westphal. After Tuerck,[1] Gulliver and Joffe had previously, in certain cases, demonstrated chronic disease of the posterior portion of the spinal cord ; Westphal's investigations taught that certain forms of paralysis uniformly correspond to certain pathological changes in the cord, and other forms at least very often to other changes.

The first of the two groups, besides the other features of dementia paralytica, presents clinically and anatomically the symptoms of tabes dorsalis, or *gray degeneration of the posterior columns.* On making a transverse section of the hardened cord, the posterior columns show few or no sections of nerve-fibres, and their place is taken by a connective-tissue substance. In the cervical region, Goll's cuneiform columns are especially affected ; in the dorsal and lumbar regions, however, the entire area of the posterior columns is involved. In fresh preparations numerous granular fat-cells and corpora amylacea are found. This change can be followed upwards only to the beginning of the fourth ventricle.

The other group, the so-called *granular-cell myelitis*, is characterized first by the presence of a very large number of granular fat-cells in the postero-lateral columns, then by increase of the interstitial connective-tissue, so that the septa and septula of the spinal cord are greatly thickened, and the fine meshes surrounding the nerve-fibres seem transformed into a thick network filled with knot-like structures.

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[1] Sitzungsber. der math. physikal. Klasse der Wiener Academie, 51, 53, 56.

The granular cells are found especially in the vicinity of the vessels, and according to L. Meyer arise from their walls; according to Odler, however, from the septa and septula in which they are found.

This change can be followed through the medulla oblongata and the pons, even into the external parts of the cerebral peduncle; but in the cerebrum itself it has not as yet been recognized. It is characterized clinically by an awkward, helpless gait, and in advanced cases by diminution of strength in the limbs.

Both processes, viz., gray degeneration of the posterior columns and granular-cell myelitis of the lateral columns, may exist together.

When forming a judgment concerning pathological changes, especially in the spinal cord, it must not be forgotten that ordinary inspection alone of fresh preparations proves nothing. First, preparations must be made by "teasing," after small portions of the substance have been macerated in a solution of bichromate of potassa, one part in one thousand, for twenty-four hours (the next twenty-four hours in a solution two parts in one thousand). Then the organ is to be hardened in a solution of the bichromate, of gradually increasing strength, from one to six per cent. By this means the diseased parts, which, when fresh, sometimes seem normal, become colored a clear yellow, and so attract attention. Then follows the examination of sections. Alcohol dissolves the fat, and hence cannot usually be employed for hardening.

The *peripheral cerebral nerves* rarely show any palpable changes; though occasionally there is gray degeneration of the nerves of higher sensation, especially the olfactory and optic (Westphal, Schuele, Magnan, Mierzejewsky).

The *posterior roots of the spinal cord*, however, in all cases which are complicated with gray degeneration of the posterior columns, are likewise affected with gray degeneration, sclerosis, corresponding to the changes found in simple tabes dorsalis.

Symptomatology.

The signs of general paralysis are divisible into two groups, the mental symptoms, and those of a different nature, especially motor disturbances. The *succession* in which these symptoms appear, is not constant: sometimes the mental changes appear first; at others the first, excepting prodromal symptoms, are some other anomalies of sensation or motion, or mental and corporeal symptoms may appear together. A certain class of patients, finally, years before the appearance of mental disturbance, show all the signs of tabes, the regular lancinating pains, anæsthesia, disturbance of co-ordination, and vesical troubles. A correct judgment in regard to the prodromal symptoms is, as we shall see, of the greatest importance, especially as these not rarely precede for years the final outbreak of the disease. Besides attacks of *dizziness*, which are quickly over, and *headache*, more or less severe, with morning exacerbations, as are found in many cerebral diseases, there appears unusual *irritability* of character, such as has not been previously noticed in the patient. Joined to this, at an early stage, is a greater or less degree of *weakness of memory* for events and one's own acts in the immediate past, while that for events of earlier life is remarkably well preserved; absent-mindedness, forgetfulness of hat, cane, pocket handkerchief, etc., sometimes are the first symptoms noticed. At other times it is a slight *trembling of the facial muscles* which attracts the physician's attention, especially of those muscles which surround the mouth, which occurs either spontaneously or when other facial muscles are in action. It must not be expected that the patients will mention this symptom; for it is, as I have found, unknown to them, even when there is not enough mental weakness to account therefor. Even when the patients are told of it, they recognize the fact only when they see it in a mirror. At the same time with this symptom, or even previously—as a rule, not separated from it by any great interval of time—*speech* is changed; it becomes nasal, or the tongue is moved with less certainty and energy, so that words are brought out with difficulty; especially are certain sounds, the labials and sibilants, imperfectly or stammeringly enunciated. This peculiarity of speech may be best compared to that of one slightly intoxicated. At this time, already, a slight disturbance in the *motions of the tongue* is seen, slight deviation, or fibrillary or general trembling. The *character of the voice* is also changed, so that, for example, a tenor becomes a bass. Finally, a difference in the size and reaction of the *pupils* may be noticed at an early stage; but it must be remembered that this symptom may be found in persons disposed to other nervous affections, and is not found in at least fifty per cent. of the patients. Under all circumstances the coincidence of several of these symptoms, especially disturbance of speech, trembling of the facial muscles, a loss of memory, though only slight, should lead the physician to adopt a very circumspect course.

The *change in the mental functions*, apart from the very constant prodromes already mentioned, shows a character and course which vary within wide limits. Yet it must be remembered that, in most cases, the whole exhibition of mental phenomena has something so characteristic that the experienced observer recognizes the nature of the mental disturbance from the first word the patient utters, and even those less experienced may see their way clearly.

The view originally promulgated by Bayle—that the *nature of the delusions*, in so far as they imply exaggerated ideas of the patient's own importance or greatness, is characteristic of the disease—has, properly enough, been disputed. Yet ideas of self-grandeur are so frequent in this disease, and occur in such astonishing forms, that they give it a peculiar impress; while one suffering from simple maniacal exaltation or madness will often give expression to insane ideas, which, to a non-medical person, or one not informed of the circumstances, will scarcely seem singular, the senselessness, the utter absurdity of the delusions of the general paralytic, so soon as they take their typical form, cannot fail to attract the attention even of the laity. The insane person, for example, perhaps imagines himself the son of some man of high rank, and as such either expects great riches or considers that he has a claim to them; or he is very clever—

has discovered perpetual motion—will soon give a public lecture—will benefit the world by his ideas. There is always a certain method in his madness, a method devised with wonderful acuteness, and which may be a very convincing evidence to the laity of the mental soundness of the insane.

The general paralytic, on the other hand, has one thousand—yes, a million horses, if a thousand are not sufficient; he has a thousand million dollars; reaches with his hand to the moon, immediately acknowledges that his arm is not long enough for that, but immediately after reaches again to the moon, without being disturbed or made angry by the contradiction which is evident to everybody, and has been proved to him. Thus it is possible easily to draw from this patient any desired delusion, to excite the idea in him; words have evidently lost their meaning for him; he employs them in a conventional, routine way, somewhat as one takes off his hat—without giving a thought to the action. The general sense of prosperity, which is peculiar to these patients, causes them to use the most exaggerated terms; but what they say and do is immediately forgotten again, so that the most contradictory things do not appear so to them, and the adjective may deprive the following noun of all sense.

The demeanor of the paralytic—peculiar from the loss of all power of judgment—acquires still more the character of the most idiotic silliness from the weakness of memory which is found at the very beginning of the disease.

The following is quite a characteristic example:

One of my patients regularly answered the question how many ears he had, by saying he had a great many. If asked, "How many, then," he began to count them, but counted his eyes with them, and always began anew, until he finally stopped at some number. Beautiful, also, is the title of a poem with which one of my paralytics surprised me one morning: "The fight with the *two* dragons, poem by Schiller von Ottmar." Such was his poetical pseudonym.

The *natural affections* show the same character of weakness which gives the disease its peculiar coloring. Notwithstanding lively motor manifestations, the excitation of the feelings is neither deep nor lasting. Generally the patients do not in the most remote degree trouble themselves about their families and their fortunes, or they forget the existence of all others in their excessive care for one member of their family. At the same time it is noticeable how, in paralytics, the *possibility* of being touched by emotions of affection sometimes long survives the death of their other mental faculties. The good, though fleeting, effect of a friendly word or hand-grasp is perceived even in the late stages.

As a matter of course the mental changes, in the majority of cases, do not suddenly appear in such a gross form, but there is a *transition*, a *development by stages*, which inspires a certain anxiety in the family friends, but which they are disposed to explain in any other rather than the true way. Herein lies especially the social danger to be feared from paralytics. They often destroy in a short time the well-being, the very subsistence of their families, and even draw outsiders into the misfortune, because, being already filled with exaggerated notions, they have lost every idea of the worth of money and the significance of their own personality.

In other cases the patients become especially dangerous from sudden *outbursts of anger* of such intensity and blindness as can be matched only in the epileptoid or post-epileptic condition. Aroused by a contradiction, or by some other injudicious treatment—often, too, without any evident cause—they fall into a blind rage, in which, in an insane way, they threaten those about them, and even themselves, with destruction. Thus one of my patients made a murderous attack upon his wife because she had left the window open. Such attacks, more or less outspoken, sometimes having only the external form of slight excitation, occur from time to time during the further course of the disease and give a certain variety to the monotony of the delirium, which drags along through years, becoming continually more imbecile. During these periods of exaltation the motor disturbance previously existing generally disappears, wholly or in part, the patients are unusually powerful, and therefore so much the more dangerous.

In other cases the initial stage of excitement does not cease, but persists until death, which then occurs early. Such cases may easily be mistaken for acute delirium. Again, instances occur where the maniacal excitation lasts for weeks, patients tearing their clothes, besmearing themselves with excrement, etc., and then ceases while absolute dementia suddenly sets in. There is no longer the slightest sign of an idea—indeed, even the understanding of spoken words is reduced to a minimum.

According to L. Meyer, these attacks are accompanied with increase of temperature, and in so far are to be considered as the expression of the exacerbation of an already existing chronic meningitis. But Westphal has shown that the increase of temperature, if it be really present, is explained by accessory or intercurrent affections, and Meyer himself does not seem to attach the same value to this part of his opinion as formerly.

Although we have emphasized the fact that a false conviction of being well off, an insane idea of one's own grandeur, and a condition of excitation susceptible of excessive increase, form the well-marked features of the ordinary form of the disease, yet these symptoms are found neither in all patients nor in all stages of the disease; but in a certain number of cases we meet with depressing, hypochondriacal, melancholic states of mind, which, it is proper to state, may, together with weakness of memory, not rarely form the initial mental symptoms. These ideas, too, while possessing a certain degree of uniformity, are also characterized by unusual weakness of judgment, which lies at their foundation. The patients imagine that they are three-cornered, that they are sewed up all over, or make similar statements over which, as in many other mental diseases, there can be no discussion.

These conditions of excitation and depression by no means mutually exclude each other, but rather, in many cases, are interchangeable, so that certain authorities find a comparison with the so-called *folie circulaire* justified. After a period of hypochondria the patients pass through one of mania, after which there follows again one of hypochondrio-melancholia. In other cases the ideas of grandeur are suddenly interrupted by simple absurd ideas of a depressing nature; or the patients, without any reason, suddenly fall to crying and complaining distractedly and idiotically for some hours. Other patients again fall into a state of melancholy, for a long or shorter time, for the best of reasons, viz., because they still have judgment enough to appreciate this or that symptom which they notice, as the sign of a serious disease.

Delusions may be entirely wanting, or at least may be absent for a very long time—a fact which has led to the designation of *paralysis without insanity.* If intellectual weakness is not to be considered as a form of insanity, this designation is certainly correct, as I myself can show by numerous examples. Nevertheless, the psychosis as such is characterized by the very signs of defect or weakness heretofore mentioned, so that the above view must be looked upon as an error only calculated to cause confusion. Such patients, it is true, do sometimes gradually pass away with increasing signs of imbecility, and without ever having delusions; but sometimes the last stage is still marked by the characteristic delirium of imagined grandeur.

The view has been many times expressed that *absence of the consciousness of disease* is pathognomonic of general paralysis. This may prove true in the stage of marked dementia, inasmuch as patients, then, are no longer in a condition to recognize and form a judgment upon their own state; in asylums they do not notice the quality of their surroundings, nor even the change in the character of their ordinary clothing where a uniform dress is required. During the earlier stage of maniacal exaltation, too, they frequently enough have no sense of ill health, but boast of their robust health, declare that they are as strong as ten men, and require a thousand wives to gratify their sexual power. Much the same state of things, however, is found in many other forms of insanity. The madman, the partially deranged person, for example, is much more obstinate in the support of his imaginary superiority. In general paralysis, again, only the senseless exaggeration with which the patient speaks of his good health is characteristic. In other cases, however, and only too frequently, the patients recognize very well what their mental and bodily condition is, and from this knowledge spring, as above mentioned, the hypochondriac and melancholic ideas, to which, moreover, some special coloring may be lent by peculiar bodily sensations experienced by the patient. Equally curious delusions, not melancholic in character, may also occur, based on the same sort of sensations (illusions). Thus one of my patients thought that he always carried two calves on his naked body, into whose mouths Emmenthal buttermilk constantly flowed out of his breast through two tubes.

It seems that *illusions* in the domain of general sensation are by far the most frequent specimens of sensory deception in dementia paralytica. If hallucinations of sight and sound are not entirely wanting, as has been asserted (Huppert), they are decidedly insignificant as compared with what occur in other forms of insanity. A patient in my asylum, a resident of Prussia, during a remission in his symptoms, confided to me that he must have been very sick, for he had had hallucinations. A voice, which seemingly came from heaven, had continually assured him that he had become a count and a Russian state councillor.

Special mention must be made, on account of its forensic importance, of the very frequent occurrence of *kleptomania*—if this word is allowable—in general paralysis. These patients pocket unnecessary articles, or do not indeed pocket them, but carry them away openly, because they believe they are making use of their own property, or because they pocket everything which pleases them, without giving it another thought. Not infrequently they are condemned for theft, and their punishment is made more severe because of their shameless lying, because they deny the act, long since forgotten by them, or regard it with supreme indifference.

If we turn now to the further course of the *motor changes*, we must remember, in the first place, the two forms of motor disturbance which were more clearly described by Westphal, as was mentioned above. The one, which pathologically resembles tabes dorsalis, has also a clinical likeness to that affection, excepting that a peculiar expression is given to the phenomena by the disturbances in the facial and hypoglossal nerves, as well as by the mental symptoms. Staggering when the eyes are shut, a jerking, uncertain gait, difficulty of turning round, a peculiar trembling, jerky handwriting—in a word, disturbances of co-ordination, first and especially in the lower, then also in the upper extremities, amounting sometimes to entire inability to use the limbs, are common to both diseases. To make the resemblance between the two still more complete, both may be accompanied with incontinence or retention of urine and fæces, finer and coarser disturbances of sensation, especially in the chest and legs; finally, with amaurosis and more ephemeral paralysis of the ocular muscles.

The other form, which has the anatomical characters of granular-cell myelitis, is accompanied by rather a shuffling, awkward, helpless gait, unsteadiness on attempting to turn quickly, but no increased swaying of the body when the eyes are shut.

Real, complete, and *persistent paralysis* of all the muscles, as might be expected from the name "paralysis," is not seldom wanting in cases of the disease which run a rapid course, or appears only towards the end. Yet a more or less complete, persistent, unilateral paralysis of the facial nerve is often noticed.

On the other hand, *apoplectic attacks*, with the well-known signs of cerebral apoplexy, rarely fail to occur—often, indeed, being the first sign of our disease. They leave behind a more or less well-marked and extensive hemiplegia, which, however, almost always disappears again with wonderful rapidity—at least so far as concerns motion—while the intelligence, after every such attack, degenerates so much the more quickly. Sometimes slight hemiparesis persists even to the end of life. In the place of these apoplectic attacks involving the motor functions, I have repeatedly seen quite analogous phenomena affecting the sensory functions, sudden attacks of vertigo, after which there remain abnormal sensations of tension, of a mask before the face; also slight loss of the sense of touch, which can be recognized objectively and quickly disappears.

The attack is often combined with unilateral or general *epileptiform* spasms, in which, occasionally, death occurs. Another time the attack is apparently of less consequence; it takes the form of the petit mal, of a slight attack of faintness, or complete loss of consciousness. Yet this form has also a serious significance, pointing to a more rapid course of the disease. We must here repeat that in the vast majority of cases the autopsy warrants no conclusion as to the anatomical cause of these phenomena occurring so suddenly, and not rarely with great severity, and that especially the proof of gross cerebral changes is extremely rare.

During these attacks there is often a very rapid increase of temperature. Westphal has endeavored to refer this to manifest or latent disease of the respiratory organs. However, I must say that, so far as I have seen, this explanation seems suited to only a part of the cases, and that I do not hesitate to allow "unknown nervous influences" to pass as an important factor.

Persistent and extreme *anæsthesia* finally occurs in both (of Westphal's) forms of paralysis. We mentioned this when speaking of cases complicated with tabes, yet it may be recognized also in the advanced stages of other forms, notwithstanding the extreme dementia. Not only do these patients not pay the least attention to deep pricks with a needle, but they also bear serious surgical operations or frightful self-mutilations without giving a sign of pain.

The number of theories which have been advanced in regard to general paralysis are almost as great as the number of observers of this disease. I will, in what follows, limit myself to collecting a few fixed facts and views which are defended by a large number of observers.

In considering the anatomical changes as given above, we found very manifold diseases of the brain and its membranes, and if a complete enumeration were made the number would have to be considerably increased. Now it is quite evident that the diseases of the membranes and bones can claim only a secondary significance. To be sure, the destructive process may often enough take its origin from them, especially from the pia mater; but, after all, at the last it is always the *brain itself* whose alteration causes the symptoms.

In fact, the final cerebral atrophy, which, as we have already said, is by far the most common change, teaches us that some inflammatory process must have existed previously, and other changes seem also to show fresh traces of it.

Finally, anatomical changes in the *spinal cord* are found so frequently and in so characteristic a form that their intimate connection with the cerebral disease must be acknowledged.

What now is the nature of this connection? That is one of the first and most important questions.

We have seen that one class of cases not only runs its course accompanied with spinal symptoms, but that the latter may for years precede the cerebral symptoms. In these cases the spinal cord shows post-mortem changes entirely analogous to those usually found in ascending degeneration.

Now it is possible that there may be ascending degeneration commencing in the spinal cord, and gradually working its way by continuity to the cerebral cortex. According to Huguenin, in certain individual cases the gray degeneration of the posterior columns has been followed through the upper pyramidal decussation into the cerebral crura. The same author also believes that a rich deposit of cellular elements (emigrated lymph-corpuscles), which he could recognize in the internal capsule and the corona radiata, bore some relation to the morbid process. If these observations should increase in number and prove confirmatory, the above supposition would, at all events, have the probabilities in its favor.

Meanwhile, it is just as possible that the parts of the central system functionally related to each other, either congenitally, or in consequence of some previous injury, suffer from a similar predisposition to disease, to which one part in one case, the other portion in another case, yields more easily and quickly; and, finally, it may be supposed that disease of the virtual continuations and endings of certain tracts may occur without involving their entire continuity. It is, indeed, not incredible that the loss of certain normal irritations, or the addition of certain pathological irritations, might be absolutely injurious to certain portions, while in others only comparatively slight or perhaps no disturbance might be excited.

In the *second class* of cases (granular-cell myelitis) the disease of the spinal cord follows the same course as the descending degeneration, which has been known to us since Tuerck's investigations as following lesions of the radiations of the basis cruris cerebri, especially the inner capsule, so that in these cases the supposition of a centrifugal extension is sufficiently justified. Now it is true, as has already been repeatedly stated, that we find no *foci* of degeneration of the nerve-tissue (or, at least, that such is the very rare exception); at the same time it cannot be denied that *not very extensive*, *more delicate* diseases of the cortex may produce similar effects. Smaller artificial lesions of the cortex seem, indeed, not to cause secondary degeneration of the cerebral crura and spinal cord. Yet this matter is only just beginning to be worked up. We may soon expect an important publication from Gudden on this point, and I myself have been for a long time employed in similar investigations.

Here, too, we must also for the present leave room for the other possibilities mentioned above. In a pathogenetic point of view, the brain and spinal cord cannot be separated; but it must be clearly maintained that over-use as well as other injurious influences are felt in *both* spheres, though their traces are more easily found sometimes in the one, and again in the other.

And this seems to answer, in accordance with our present knowledge, the question, *from which organ, from brain or from spinal cord, do the motor troubles arise in general paralysis.* It would be one-sided to attempt to give a general answer applicable to all cases; each separate case will have to be judged by its own peculiarities.

The possibility of an accurate localization of the motor disturbances in the brain will presently be discussed.

The peculiarities of the mental symptoms in general paralysis have been objects of much speculation. We cannot recognize in these speculations anything more than the expression of the private views of the authors referred to. The different psychoses are not susceptible of a separate consideration, but they must all be treated of together. At present, however, we are still wanting in the necessary material for this purpose.

Only two facts here seem certain. Firstly, during the *condition* of *mental* and *motor excitement* there must be greater activity of the cerebral centres, corresponding to which must be an increased waste of material, and consequently *greater supply of nutrient substance—blood.* For, as more manifestations of force appear, it must be that more force has been used up. Quality has nothing at all to do in the matter; it is merely a question of amount. But this holds good of all insane excitement. Nothing specific can be deduced, therefrom, with regard to general paralysis.

Secondly, the weakness; the mental poverty of the later stages is doubtless to be explained by the cerebral atrophy. But in this, too, there is nothing specific; for in other chronic forms of insanity exactly the same atrophy is found. Certainly, it might be maintained with some truth that there is not any very great difference in mental function when a paralytic woman imagines that she has a thousand golden dresses, and a crazy woman states that she is empress of the whole world, the chaplain of the hospital is her husband, and he must be required to give her a child which will be the new Christ. Both are certainly the products of a dementia accompanied with a feeling of perfect contentment; and if special varieties are claimed for general paralysis, it can be replied that they are not constant. In fact, a further consideration of such reflections would only lead to useless discussion.

We shall make a little more progress when we inquire into the physiological basis of the cerebral motor disturbances in general paralysis. In this there is truly something specific to this psychosis alone. The other varieties of weakness, accompanied with cerebral atrophy, show no analogous symptoms dependent thereon.

If we exclude entirely those cases in which the beginning of the affection, and consequently also the beginning of the motor disturbance, is to be found in the spinal cord, there still remain a considerable number which certainly begin in the brain. But if we ask what there is specific in these which separates them from the other forms of insanity, the view involuntarily intrudes itself upon us that this lies less in the nature than in the locality of the affection. Not that I would say that in general paralysis a certain area of the brain is affected and the rest is free. I am not at all of this opinion. But if we see serious motor disturbance set in in this disease, as the result of a pathological process to which we will and can ascribe no specific nature, but which does not induce total destruction of the cerebral mass, and if we miss these motor disturbances in other disorganizations seemingly analogous, it seems at least probable that in the cases first mentioned there is an intense action of that minuter morbid process in some restricted locality which has something to do with motion.

Such localities have been proved to exist in the cerebrum by the experiments made by myself,[1] and in part by Fritsch and myself on different species of animals; also by the experiments of Nothnagel[2] and others.

If we irritated certain narrowly circumscribed spots on the cerebral cortex with weak electric currents, twitchings occurred on the opposite side of the body, sometimes in the extremities, sometimes in the head. As a result of such irritations, epileptiform attacks were also developed, which bore the closest resemblance to the attacks of paralytics.

But if we destroyed the corresponding portions of the cortex, disturbance of motion immediately followed, which had the greatest similarity with the symptoms pathognomonic of gray degeneration of the posterior columns, and which are seen often enough in general paralysis. The animals slipped about on the smooth floor, placed their legs unsteadily, and allowed themselves to be put in the most uncomfortable postures without noticing it.

In the lower animals these spots lie rather far forward. In apes they are in the anterior central convolution, so that the entire frontal lobe still rises anteriorly to them. A number of observations favor the opinion that in man also there is an identical mechanism in the anterior central convolution, but there is not so much certainty of this as in animals.

It may not be too venturesome if I express the opinion that pathological changes of any kind which affect this area in the human brain may produce similar symptoms to the artificial lesions in animals; but I repeat it, the notion must not be favored that in general paralysis the lesion is very circumscribed, for it is certain that the destruction attacks large regions.

The above-mentioned post-mortem appearances are very strong evidence in favor of this view, and I must say that the reports of foreign authors, especially those that appeared before the publication of my experiments, prove the same more than do my own pathological observations, inasmuch as they show more constant changes. Other authors, much more often than myself, found the anterior part of the brain adherent to the membranes, or covered with a large amount of serum. Perhaps the extent of my observations may have been too small.

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[1] *Hitzig*, Untersuchungen über das Gehirn. Berlin, 1874.

[2] Virchow's Arch.

While I am, therefore, under greater obligation, for this very reason, to express myself as guardedly as possible, yet, on the other hand, an entire ignoring of my own experimental results would seem as forced as it would be unjustifiable. In conclusion, mention here must be made of the view contended for by Meynert, that the peculiar weakness of memory noticed even in the initial stage of general paralysis is referable to an early invasion of the frontal lobe. This view is supported by Meynert's anatomical investigations, as well as by the fact that in the gradual succession from the primates up to man the frontal lobe continuously increases in size.

But, on the other hand, we must not forget that entire unanimity by no means exists in regard to the preponderance of pathological changes in the frontal lobes, so that the early and even later alterations in very many cases must be more of a functional than of a destructive nature; for such considerable and lasting intermissions of the disease as we have referred to when considering its course, are, indeed, well-nigh impossible, without an almost complete *restitutio in integrum.*

Diagnosis.

The *diagnosis* of dementia paralytica is uncommonly easy in well-marked cases. The sense of *bien être*, the ideas of grandeur, the trembling of the facial muscles, with possibly a facial paresis, and the stammering speech, are all signs which quite frequently inspire a sad certainty during the first few minutes of the examination.

But the diagnosis may be more difficult, and sometimes impossible, when a case is seen during the condition of exaltation above described, unaccompanied by any history. Especially during the course of alcoholism, attacks occur which resemble outbreaks of madness as closely as two eggs resemble each other, but which justify a very different prognosis. Indeed, a longer observation may sometimes not show the difference, for, when the patients have become quiet, they still show slight paralytic symptoms in the face and tongue; they tremble and stammer somewhat still, and exhibit a greater or less defect of intelligence. Now this may be a case of chronic alcoholism in the above-mentioned stage, as well as a case of general paralysis in its stage of remission. Since alcoholic excesses play a large rôle in paralysis, the etiology does not prove much.

A valuable means of diagnosis is found in *testing the memory.* A patient who clearly states events in the distant past, and leaves enormous gaps in those of the last few days, is not likely to be anything else than a paralytic.

Again, the presence of symptoms of tabes weighs heavily in favor of the diagnosis of general paralysis. At the same time every alienist will occasionally dismiss patients of this kind without being able to give a positive diagnosis, and will rather depend upon their return to settle the question.

Other conditions of *maniacal agitation, with derangement of intellect,* often give rise to long-continued doubt, especially if there is also paresis of the facial nerve. Here, too, alcohol sometimes plays a prominent part, even where the patient should not be called a drinking man. If the derangement of mind is wanting, it is much easier to come to a correct conclusion, guided by the presence or absence of weak paralytic delirium. In that case the mistake most easily made is in confounding this condition with *senile dementia.* It is, indeed, very probable that the statistics of some authors, who have noticed general paralysis with unusual frequency in advanced age, have been very much influenced by such mistakes, which cannot, indeed, be very severely blamed.

To be perfectly candid, we must confess that there is not a single symptom which is so pathognomonic of general paralysis that it may not be found also in senile dementia, and, excepting the peculiar weakness of memory, in alcoholism also; and even the grouping of the symptoms as a whole may in the two latter diseases be such as to afford an entire analogy with undoubted cases of general paralysis. In senile dementia the course of the affection itself often enough furnishes no definite conclusion, while alcoholism certainly affords a very much more favorable prognosis.

Now it would certainly seem as if a study of the pathological anatomy of these affections would eventually furnish an explanation of this clinical resemblance. Especial study should be devoted to the nature of the process under which the cerebral atrophy takes place, which is peculiar to all these affections.

Only those cases can with certainty be considered as senile dementia in which advanced age, want of motor disturbances, or the presence of very marked hemiplegia, and, finally, absence of the extravagant delirium of grandeur, are all found associated. The delirium of old age is, as a rule, almost invariably of a more childish nature, while it is concerned with the most diverse subjects. Besides, epileptiform attacks almost never occur in the dementia of age, and the apoplectic attacks of these old men are accompanied with serious permanent paralyses, if they do not end in death. Finally, the course of senile dementia is regular, less broken by remissions and intermissions. Nevertheless, that is an uncertain criterion, and the same may be said to a still higher degree of all the other diagnostic points mentioned above.

Space forbids us to notice any further the numerous other affections with which general paralysis may be confounded, and we must for this purpose refer to special works and journal articles (*e.g.*, v. Krafft-Ebing, loc. cit.).

We only remark, in addition, that bulbar paralysis, gray degeneration of the posterior columns of the cord, multiple centres of encephalomalacia, and finally, epilepsy, are the conditions most frequently confounded with this affection, whenever psychical symptoms are added to one of these diseases.

Course, Duration, Termination, Prognosis.

From data given above it is evident that the duration and course of general paralysis must be very various. At one time the disease runs its course comparatively rapidly, at another it extends over a series of years.

As a general rule the cases that progress rapidly are rare, as would be inferred from the fact that L. Meyer, during a long and extended practice, could select only twenty such for his work, so often quoted (Die Pathologische Anatomie, u. s. w.). In these the disease seems to have ended fatally in a period varying from a few weeks to one year. However, the greatest uncertainty attaches to these data. One must himself have experienced how the public refuses to acknowledge even extreme imbecility as a mental disease, in order to understand the great difficulty of fixing the beginning of these diseases with even approximate certainty.

Naturally the duration of the disease can be better calculated if only the time of confinement in an asylum is considered. Here, indeed, most authors agree tolerably well. Most patients seem to die between the fifteenth and thirtieth months after admission. This certainly implies that the patients come into the asylum comparatively early, and that the external surroundings of the asylum, nourishment, attendance, etc., are favorable, otherwise the duration of life could not be placed so high.

On the other hand, exceptional cases are known in which the disease has continued more than ten years before the fatal termination. If the commencement of the disease were dated from the first symptoms of the tabes of the spine—that is, from the appearance of the lancinating pain and the affection of the bladder, such cases would no longer be rare.

Most cases of general paralysis run their course with all the individual symptoms becoming gradually worse and worse, until apoplectic attacks occur, in consequence of which the patient goes down hill by jumps. But it must not be forgotten that, as a rule, by no means the entire amount of deterioration following an apoplectic attack remains permanently, but that almost always a large part of the ground lost is regained between the attacks.

Gradually the expression of countenance becomes more vacant, more senseless, more staring, more flaccid; the muscles of the face fall into irregular vibrations upon every attempt to speak, or upon any mental emotion, or even spontaneously; speech finally becomes so stammering that the patient can no longer be understood. Coherent conversation had already previously become more and more rare, and now entire groups of words begin to disappear, so that the mental deficiency goes hand in hand with the mechanical. Often ceaseless smacking of the lips, or gritting of the teeth, is noticed. The other voluntary motions likewise become constantly more uncertain, less frequent, and weaker. Therefore, before long, patients get into the habit of sitting motionless for hours at a time, or they become bedridden, pass their fæces and urine in the bed, and, notwithstanding the greatest attention and cleanliness, develop bed-sores, which may endanger their lives. Sometimes contractions still further interfere with motion.

The *general nutrition*, generally, also suffers rapidly, so that before death there is great emaciation. Occasionally, it is true, just the opposite happens: the patients become unnaturally fleshy, have a prominent abdomen; a thick, full face, which then looks especially imbecile.

Death frequently takes place during a paralytic attack, or somewhat unexpectedly, from pneumonia, caused by the passage of food into the bronchial tubes. In other cases an acute lung disease occurs without any known cause, or pulmonary tuberculosis, bed-sores, bladder or kidney disease set in—a mournful termination to the sad spectacle.

In a small proportion of the cases the course is different. Suddenly, at a time when the excessive imbecility has caused every hope to disappear, an improvement sets in, most unexpected to the inexperienced, and very difficult to explain. The memory, the power of judgment and speech, improve sometimes with remarkable rapidity, to such a degree that the patients become capable of returning to their former course of life.

With reference to prognosis, there is every reason for the physician to look upon this remission of the disease with the greatest distrust, and especially to guard himself from giving the friends positive hope, or seeming to claim the credit of a cure.

Cases are known, it is true, wherein the remission has developed into lasting, or at least, long-continued recovery. Nasse[1] has collected the published facts on this subject up to the year

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[1] Zur Diagnose und Prognose der allgemeinen fortschreitenden Paralyse der Irren. Irrenfreund, 1870. No. 7, p. 97.

1870, and according to these there can be no doubt that such turns in cases of well-marked general paralysis do occur. Schuele,[1] among others, has lately reported a similar case, which he followed for five years. Intercurrent acute diseases (typhus fever, small-pox, suppuration) seem occasionally to have a favorable influence, as in other psychoses. Yet, it is very noticeable that every author who reports recoveries, has, quite properly, found it necessary to guard his diagnosis, so far as possible, against doubt. For, in very truth, *these cases of recovery from general paralysis are among the most exceptional.* They occur just often enough, however, to prevent a physician from giving an unfavorable prognosis with too great certainty.

As a rule, it remains true that the doom of the general paralytic is sealed. An effort has often been made to decide in regard to the longer or shorter duration of a remission, from the presence or absence of one or another symptom. Hitherto these efforts have produced no acknowledged results. It will be much the safest to suspect a speedy exacerbation in the case of every patient who exhibits newly acquired symptoms of the disease during its progress ; and the more symptoms he shows, the more reason for suspecting this. Besides, a moderate degree of mental weakness may escape the notice even of an expert, if he has not known the patient previously, if he is not acquainted with his circumstances, and has to judge of the case perhaps out of the asylum.

These considerations, as well as the circumstances of the prodromal stage, may place the physician in a very unpleasant position before a court, and hence in each individual case should be most carefully weighed.[2]

Treatment.

The therapeutics of progressive paralysis is rich in remedies as it is poor in results. Perhaps rather more might be accomplished, if the relation of the public and of physicians to asylums for *curing* the insane should change. A singularly large number of members of the profession still mistake their purpose, regarding them as asylums for *caring* for the insane. Therefore they do not advise the patient to be taken to an asylum until it is absolutely impossible to look after him privately, while the physician is in duty bound to be guided only by the consideration how and where the patient can be healed most quickly.

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[1] Beiträge, etc., Allgem. Zeitschr. f. Psych. Bd. XXXII.

[2] *Solbrig*, Allgem. Zeitschr. f. Psych., 1865. Bd XXII., p. 397.

But if the general paralytic is treated at home, his chances of recovery are decidedly less than when *at an early stage he is placed in a well-conducted asylum, furnished with large gardens.* It is very seldom, indeed, that the laity, or even the family physician, understands how rightly to manage the patients. They are irritated by contradiction and restraint, or because an attempt is made to argue them out of their delusions, eccentric plans, etc. They are excited by diversions, travels, and other improper methods of treatment, until soon the disease takes a form which, on the one hand, renders a seclusion of the patient necessary, and, on the other hand, this seclusion begins under much less favorable conditions.

The first requisite for successful treatment, which cannot be neglected, is physical and mental *rest*, under continuous skilled oversight, and this cannot be found in full measure excepting at an asylum. Here the patient should have the most free enjoyment of *fresh air*, and easily digested, yet rich and strengthening *nourishment*. *Wine* in moderate amount at meal times is usually well borne, but taken in larger quantity it is decidedly injurious. *Tobacco* is entirely unfit for him.

Apart from the interests of the patient, the welfare of his family and of strangers requires that he should be restrained from working mischief as early as possible. It has been already stated how easily, in the initial stage of the paralysis, patients may be possessed by an overmastering idea of their own personal merit, of their ability and property, and so be led into the most senseless transactions. They throw their money about freely, make ridiculous contracts, marry, commit indecent or other brutal outrages, etc. The weak-mindedness of the patients leads to other misfortunes in the fullest significance of the word, since they occasion the wildest confusion in their own business and in that of others before their neighbors, not understanding the subject, suspect anything wrong.

In the initial stage of the disease I have seen undoubtedly good effects from the action of *galvanism* upon the medulla oblongata and upper part of the spinal cord, but this good effect was only temporary. The disturbance of speech especially improved during the sitting. Similar statements are made by other authors, as Newth[1] and Arndt[2] seem to have had a similar experience. Iodide of potassium and cool sponge-baths seem to me also occasionally to have been of temporary value.

On the other hand, all harsh or debilitating treatment is to be most carefully avoided, especially the cold-water cure proper; yet, notwithstanding all warnings, the misuse of this continues to thrive; bleeding and mercurial preparations may be included in the same category.

The latter owe their reputation, doubtful as it is, to those cases depending upon a syphilitic taint. Here they may sometimes be of value, though the improvement is often enough not lasting. In these cases—which should be selected with care—while special attention is paid to the nutrition, the use of mercury by inunction should be preferred to the internal exhibition of the drug. Lately the preparations of *Calabar bean* have been praised as a kind of specific. George Thompson[3] believes that in the earlier stages of general paralysis there is a general vascular spasm, basing his belief upon the results obtained by examination with the sphygmograph and the ophthalmoscope; he therefore employs this agent to cause dilatation of the vessels. Crichton Browne,[4] on the other hand, publishes two cases of *advanced* paralysis which were cured by Calabar bean. He gave one-quarter to one-third of a grain of the extract three times a day for nine months or a year. He adds no remarks to his report. My own experience with the agent is less assuring, yet the patients do not seem to have been injured by it.

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[1] The galvanic current applied in the treatment of insanity. Journ. of Ment. Sci., April, 1873, p. 79.

[2] Die Elektric. in der Psych. Arch. f. Psych. u. Nervenkr. Bd. II. 1870.

[3] On the Physiol. of Gen. Paral., etc. Journal of Ment. Sci., June, 1875, p. 579.

[4] Brit. Med. Journ., Oct. 24, 1874.

The intercurrent conditions of excitement need to be specially considered. In the first place, *prolonged lukewarm baths*—the head being kept cool—should be tried. When there is a tendency to self-injury, these are indispensable.

If these do not accomplish the desired object, recourse must be had to narcotics. Besides papaverin, narcein, and others, morphia and chloral have of late been both highly recommended and sharply attacked.

We cannot here enter upon a consideration of the different theories advanced in regard to the action of morphia any more than upon a criticism of the injurious effects ascribed to both agents, and rather refer to the literature upon this subject,[1] with the remark that the question is not yet settled.

Yet this much is certain, that, notwithstanding a few individual cases which have turned out unfortunately, both the subcutaneous injection of morphia and the internal use of chloral have not only contributed very much to produce greater quiet and order in insane asylums, but they have also been of decided benefit to the excited patients themselves. In the first place, sleep is procured, and a limit is set to the consuming rage. Next the self-inflicted injuries cease, and the various kinds of restraint used to prevent them are given up. Beginning with the smaller doses and rapidly increasing, if there is no effect, 0.01–0.2 gramme (⅙ to ⅓ of a grain) or more of a morphia salt is injected subcutaneously. The chloral is given in doses of 1–5 grammes (15 to 75 grains) and more. It is to be noticed that an effect is rarely obtained with a smaller dose than two grammes, and the larger doses should be used with special care, and never continued for a long time. The administration of a teaspoonful of bicarbonate of soda, some time before the chloral is given, is said to increase its efficacy.

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[1] *Schüle*, Dysphrenia neuralg. Carlsruhe, 1867.—*O. J. B. Wolff*, Ueber die subcutane Morphium-Ther. bei Geisteskr. Arch. f. Psych. Bd. II. 1870, p. 601.—*Knecht*, Ein Beispiel von ration. Anwend. der Subcut. Morphium-Ther. Ib. Bd. III. p. 111, 1872. —*Mendel*, Zur Therapie der Melancholie. Berl. klin. Wochenschr. 1872. Nr. 24, 25, and die Temp. der Schädelh. Virch. Arch. Bd. I. p. 12.—*Gscheidlen*, Ueber die physiol. Wirk. des essigs. Morph. Würzb. physiol. Unters. III.—On chloral hydrate: *Kelp* u. *Hansen*, Arch. f. Psych. Bd. II. Also *Arndt*. Ib. Bd. III. See the same in regard to a full bibliography concerning unpleasant concomitant symptoms and fatal cases. Also *Fürstner*. Ib. Bd. VI.

If the medium dose of one or the other of these drugs does not succeed, it is advisable to employ a combination of the two —0.1–0.2 gramme of morphia with 2.0 grammes (thirty grains) of chloral.[1]

Special care should be taken to prevent the patient from receiving injuries, so soon as the symptoms of dementia or paralysis increase to a serious degree. The evacuation of the bladder and the bowels must be secured by frequently and systematically reminding the patient thereof, and, finally, by artificial means; and the most extreme cleanliness of clothing and bed must also be maintained. Finally, these patients should be confided, in their helplessness, only to patient and intelligent attendants. Under such conditions it is often possible to prolong the lives, useless and cheerless as they are, of such helpless beings, much beyond the limits usually attained in this disease.

The author was prevented from completing the other divisions of atrophy of the brain by accepting a new position.

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[1] *Zastrowitz*, Arch. f. Psych. Bd. II., p. 514.

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